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THE EFFECT OF FITNESS AND DIET INFORMATION ON SERUM LIPIDS IN SEDENTARY ADULTS

by

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A THESIS

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ABSTRACT

The effects of a popular fitness program was studied upon 41 sedentary adults: 8 female and 4 male were in a training group (A), 10 female and 7 male were in a training plus diet information group (B), and 6 female and 6 male acted as a controls group (C). Subjects were tested prior to the beginning of the program and at the end of the classes, i.e., 12 weeks after. The following dependent variables were analyzed: body weight and fat, systolic and diastolic blood pressure, three day dietary recall, post exercise heart rate, serum lipids, serum glucose and serum uric acid.

Significant decreases in post exercise heart rate, resting systolic and diastolic blood pressure were observed in the second test in group A and B. No significant dietary alteration and no changes in percent body fat were noticeable in group A, B and C during the study. Serum analysis showed no significant changes in cholesterol, lipoproteins, lecithin cholesterol acyl transferase, glucose and uric acid.

No correlation was found between those who positively modified their serum composition and either: a better changed diet, a higher increase in activity rate, an increase in predicted VO2 max, or a higher percent body fat loss.

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I. INTRODUCTION

Our way of living has changed considerably in the last few decades. In the past, man had to do physical work, while presently in North America, the progress in technology has slotted the majority of the labor force into physically passive types of jobs. This evolution affects our welfare and fitness. In some aspects it has been very beneficial, a greater variety and availability of foods, a decrease in infectious diseases, and an increase in life expectancy. This is partly due to public investment in welfare and social security. We also have better transportation, better communication facilities, and increased productivity. However, in other ways it has been detrimental. Many people suffer from an excess of weight, malnutrition, and an increase in environment-related diseases, such as cardio-vascular and hypokinetic. Bouchard et al. (1974) has suggested ambition and motivation has decreased due to social security, and stress has increased due mainly to the lack of strategies to cope with life in our "civilized" society.

Government awareness to the need of physical activity has changed dramatically in the late sixties and seventies. New programs such as Participaction, Fitness Ontario En Forme, Shape-up of Alberta, and Kino-Quebec, show the interest in adult physical fitness programs. Similarly, professional associations and private companies have become involved in popular health programs or informational

campaigns (American Heart Association 1972, 1973, 1974, 1977; Canadian Health Foundation, 1975; Freese, 1980; Sante et Bien-Etre Social Canada, 1980; Zohman, 1979). Also, private physical fitness and diet center's fare increased, in numbers well above the population or economic growth. A parallel increase in participation of Canadian adults in the programs reflects a desire to achieve a better physical condition and protection against diseases. In 1974, Health and Welfare Canada (Lalonde) outlined the major causes of death in Canada for each age group and sex starting at age thirty for men and forty for women. Cardio-vascular disease (CVD) is a major cause of death for these age groups in both sexes.

Many factors have been identified as cardiac risk indexes. Age, sex, heredity, blood cholesterol, blood pressure, cigarette smoking, diet, body fat, lipoproteins, lack of exercise, diabetes, serum uric acid (SUA), stress, and type A personality are the most common. Several epidemiological studies and controlled experiments have shown, within their limits, that training and diet can influence cardiac risk factors. But very few studies deal with more than three factors, and it is rare that the training and diet interaction is studied. Recently, the lipoproteins have received much attention as a risk factor for CVD. The research relating lipoproteins and the individual's health status is now regarded as an important tool in assessing risk. In addition, the atherosclerotic

processes are closely related to lipoprotein metabolism.

In the aging process, man has a tendency to accumulate negative factors of the risk index, i.e., systolic and diastolic blood pressure increases (Humerfelt, 1963), blood glucose increases (Keen et al., 1965), SUA increases (Klein et al., 1973), and plasma total cholesterol and low density lipoprotein (LDL-Chol) increases. High density lipoprotein cholesterol (HDL-Chol) increases, but at a lower rate (Tamir et al., 1979). Very low density lipoprotein cholesterol (VLDL-Chol) and triglycerides (TG) increase with age up to 55 and then tend to decrease (Tamir et al., 1979; Gotto, 1978). A study of how these factors may be changed in sedentary adults, a group who tend to be high risk, may assist in the establishment of preventive programs for CVD. In addition the examination of blood changes in such a study would help in understanding the mechanisms related to these risk factors.

The purpose of this study was to monitor a physical fitness program and the diet composition of sedentary adults and to see the effect upon working capacity, body fat and selected blood parameters, including some related to lipoprotein metabolism. Secondly, this investigation proposed to distinguish the effect of the fitness program between the males and females, and how information from popular pamphlets relating to diet and cardiovascular disease can influence these adults.

Several limitations may affect the findings of this investigation other than subject selection. Control of diet and activity levels depended on the subjects' willingness to cooperate or ability to complete the study. Difficulties in methods of data collection and reliability and accuracy of measurement may also have restricted the interpretation of the obtained results.

II. REVIEW OF LITERATURE

This chapter contains a review of literature pertaining to the effect, primarily, of training, and secondly, of diet, on lipoprotein composition, lecithin:cholesterol-acyl transferase (LCAT), blood glucose, SUA, blood pressure, body weight and physical work capacity. The review is limited to studies dealing with normal subjects, i.e., hypertensive, diabetic or hyperlipoproteinemic subjects are considered to be a different population, since they have a different metabolism from normal subjects.

A. Lipoproteins and Risk Factors

Traditionally, cholesterol and TG are regarded as the main lipid determinations related to CVD. In the past decade, lipoprotein levels have received more consideration: this is due partly to a better understanding of lipoprotein metabolism, new techniques for determination and because of their high level of predictive power for CVD.

Cholesterol is well known as a risk factor. In the early 1900's investigators demonstrated that by feeding large amounts of cholesterol to rabbits, it was possible to produce arterial lesions similar to those found in human atherosclerosis (Anitschkow 1967). By observing 1900 middle aged men over a period of 20 years, Shekelle et al. (1981) supported the conclusion that lipid composition of the diet effects serum cholesterol concentration and the risk of coronary death. Criqui et al. (1980) noted that subjects who

are in the 70th percentile or above have a much higher predictive, value which is in accordance with Kesteloot (1978) who noted that cholesterol appears to be the most important discriminant between normal and pathological groups. Although these studies strongly support cholesterol determination as a powerful indicator, it should be noted that the majority of it is carried by the LDL-Chol and is also present in esterified and unesterified forms. Therefore, it may be only a part of a more complex pathway for prediction of CVD.

The evidence supporting the adverse effects of high TG in the blood is well documented (Connor, 1979; Gotto, 1978; Greten, 1978; Maccoby, 1977). But in recent years the lipoproteins have received much attention in an attempt to understand their relationship to cardio vascular-disease. The research relating lipoproteins and the health of individual is now regarded as an important tool. Lipoproteins were being related to CVD in the early fifties (Marx 1979), but received more attention in the mid-seventies. This increased concern with lipoproteins was brought about by the work of Miller and Miller (1975), who observed that the body cholesterol pool increases with decreasing HDL-Chol, but is unrelated to plasma concentration. They suggested that HDL-Chol may keep body cholesterol down by facilitating its excretion through the liver for catabolism and excretion.

HDL-Chol is a strong, though negative, risk factor at all ages (Gordon, 1977, 1979; Witzum, 1979). It has predictive power for cardio-vascular disease four times greater than LDL-Chol and eight times that of plasma cholesterol.

The prediction power is increased when HDL2-Chol is studied, whereas HDL3-Chol is not as meaningful (Miller, 1878). Though several epidemiologic surveys confirmed the inverse and independent relationship between HDL-Chol and the incidence of CVD (Gordon 1977a, 1976; Steja, 1978), the exact role of HDL-Chol metabolism is still under current investigation. The present literature suggests that HDL-Chol, through the action of LCAT, facilitates the removal of cholesterol. Via lipoprotein lipase (LPL) activity, HDL-Chol helps to prevent lipid deposits (Norby, 1978) and makes its transport possible to the liver for catabolism and excretion (Miller, 1975). Finally, it is known that free cholesterol from HDL-Chol is more rapidly incorporated into biliary cholesterol than free cholesterol from LDL-Chol (Schawartz, 1978).

LDL-Chol is a strong positive risk factor, particularly at younger ages (less than 60 years), while VLDL- Chol is more controversial (Kennel, 1971; Witztum, 1979). Evidence exists to implicate these especially when associated with elevations of LDL-Chol (Carson, 1972; Salel, 1974; Streja, 1978). LDL-Chol brings cholesterol from the liver to peripheral cells, particularly in aortic smooth muscle

cells, vascular endothelium and fibroblasts (Bondjers, 1976; Carew, 1976; Nestel, 1978), while HDL-Chol removes it. Witztum (1979) suggested the possibility that for any given level of plasma cholesterol, the ratio of LDL-Chol to HDL-Chol determines risk rather than the absolute levels of each. This ratio seems more appropriate since it is an indication of cholesterol deposit-removal.

B. Effects of training on lipoproteins

Much research has been done on human subjects and through the years the research has become more sophisticated. Present research has considered more than cholesterol or lipids, by looking at lipoprotein cholesterol levels. In this part of the review, the epidemiological studies will be looked at first, followed by the research dealing with the effect of training.

Wood (1976) investigated concentrations of fasting plasma cholesterol, TG and lipoproteins in 41 active men, ages 35-59, comparing them to a control group of the same age (Table 1). A simple calculation of LDL-Chol, HDL-Chol yielded a value 2.02 for the active group while it was 3.24 in the control. This is interesting, since in the recent literature it is this ratio that seems to be an important measure in the CVD risk index. Similar results were found by Lehtonen and Viikari (1978) when they compared lumberjacks and electricians. They observed a significantly higher HDL-Chol and a lower TG in the first group.

TABLE II.1

Plasma Chol., TG and LPP in Active Men and Control
(modified from Wood, 1976) (levels in mg %)

	TG	Chol	HDL Chol	LDL Chol	Ratio LDL/HDL
Active	70	200	64	129	2.02
Control	146	210	43	139	3.24
Ratio	.497	.899	1.488	.928	.623

Thomas (1980) did a cross-sectional study of runners to determine whether a relationship existed between total mileage run per week and the plasmal HDL-Chol. The non-runner group had a 34 mg%, the intermediate mileage (20-39 miles/week) had a 53 mg%, and the high mileage (40 miles/week) had a 69mg% HDL-Chol level. Even if there was a significant difference in the age of the groups, HDL-Chol concentrations were positively and significantly correlated with average weekly mileage run.

Krauss (1977) found similar results from HDL-Chol levels, but he also studied the HDL2-Chol and HDL3-Chol subfractions. Both subfractions were higher among runners and the HDL-Chol subfraction was more significant. The apolipoprotein A-I (apo AI), but not the apolipoprotein A-II (apo AII) has been found to be increased in well-trained men. Wood (1979) reported similar results for the HDL2-Chol subfraction, apo AI and apo AII levels.

Nikkila (1978) found a higher mean level of HDL-Chol and lipoprotein lipase (LPL) in muscle and adipose tissue among long distance runners versus control males. In combined groups of male runners and controls, there was a highly significant positive correlation (R2=.72) between serum HDL-Chol level and LPL activity of adipose tissue. Enger (1977) compared cross-country skiers with untrained subjects and the study showed a significantly higher HDL-Chol and HDL-Chol/total cholesterol ratio among the trained subjects. Cooper (1976), with a less sophisticated method of measurement, found significantly higher levels of TG and cholesterol among unfit subjects. Hartung et al., (1980a) observed a significantly higher HDL-Chol (65 mg%) in high mileage runners, while low milage were at 58 mg% and inactive subjects were at 50 mg%, but it seemed that physical activity alone was not the only explanation. Nutritional patterns may also influence marathon runners and joggers, because they were eating less meat, bacon and sausage than sedentary subjects (Hartung, 1980b).

The above studies suggest that among endurance trained subjects, the HDL-Chol, HDL2-Chol and apo AI levels are significantly higher, while the LDL-Chol is lower when they are compared to untrained subjects.

TG decreases significantly with training (Goode et al., 1966; Hunter et al., 1972; Holloszy, 1964; Lampman, 1977; Lopez, 1974; Simovelli, 1978; Hartung, 1980b) however, Gettman (1976) and Milesis (1976) found no significant

change in a sample which seemed to be normal (jail inmates). These subjects had low TG levels (range 85-111 mg%) while in other studies (Cooper, 1976) higher values were found. The discrepancy in results may be due to their diets, (Sante et Bien -Etre Social Canada, 1980) their age, which is fairly low (20-30 years) and pre-training level (Lampman, 1977).

The cholesterol level studies remain very controversial. Some studies have indicated a significant decrease in cholesterol levels following a training program (Golding, 1961; Milesis, 1974; Melish, 1974; Squires et al., 1979; Hicks et al., 1980) and others found no significant change (Holloszy, 1964; Goode et al., 1966; Lopez, 1974; Gettman, 1976). As mentioned for TG, the serum cholesterol seemed to decrease, particularly in subjects whose levels were high initially (Montoye, 1959; Cambell, 1968; Milesis, 1974), however, total serum cholesterol is not an indication of its distribution among lipoprotein fractions. HDL-Chol increases significantly as a result of training (Altekruse and Wilmore, 1973; Lopez, 1974; Squires, 1979; Farrel, 1980; Hicks, 1980; Kinsman, 1980), with best results coming from a high intensity of training (80-85% of VO2 max, 3 times/week, 20-60 min/session, for 12 weeks). LDL-Chol has been found to significantly decrease with training (Altekruse and Wilmore, 1973; Kinsman, 1980) while VLDL-Chol has also been shown to decrease (Altekruse and Wilmore), which may be strongly related to the decrease of TG in their study. Ready (1980) found no significant change in HDL-Chol and LDL-VLDL-Chol

from a training program, but when the analysis of variance made from the ratio of HDL-Chol/LDL-VLDL-Chol, the results were very significant (0.001), therefore showing that exercise may influence the lipid levels. Similar changes were observed with coronary disease patients (Hartung et al., 1981) showing an increase in HDL-Chol and HDL-Chol/total cholesterol. This study, and the others mentioned, strongly suggest that lipoprotein levels or ratios are significantly altered by exercise training in young and old individuals. Allison and Immarino (1980) observed, in spite of an increase of VO2 max in their training program, an average decrease of HDL-Chol, but they found that their subjects increased their dietary fat, which may have influenced their results.

C. Diet and prevention programs on CVD

Though the primary purpose of this study does not deal with the effect of diet on lipoproteins, the evidence supporting the need of a good diet as part of a preventive policy against CVD warrants the necessity to include a review of this area.

Many factors are considered important as a preventive measure. The eating of breakfast, fibers, refined sugar, salt alcohol, cholesterol, poly-unsaturated fatty acids (PUFA), TG, plus nibbling habits, cigarette smoking, sleeping, weight, stress and physical activity are among the most often named (Merchant, 1978; Rodnick and Bubb, 1978;

Epstein, 1979; Thomas, 1979; Wiley and Camacho, 1980; Breslow and Enstrom, 1980). The current understandings of dietary influence is strong enough to acknowledge the need for a control in food composition as a preventive measure againts CVD (Rifking et al., 1979), and international comparisons of lipid composition of the diet and CVD tend to support this (Sakai et al., 1977; Turner, 1978). Finally, Shekelle et al., (1981) in a study lasting 20 years, showed that subjects with low scores, i.e., high relative PUFA and low cholesterol intake, had a lower death rate. This study supports the idea that dietary cholesterol and PUFA may be related to atherosclerosis. The mechanism suggested was an alteration in the structure or composition of plasma lipoproteins and their metabolism, which seems to be in accordance with previous parts of this study.

Giving information to the population in order to lower the CVD risk factors has shown significant results (Arntzenius et al., 1978), even though the approach was neither agressive, nor continuous, i.e., no pressure was exerted. It was up to the subject to follow the instructions which were given at four month intervals for one year. The results were a decrease in cholesterol, systolic and diastolic blood pressure, cigarette smoking and body weight.

Dietary advice with respect to cholesterol, PUFA, and a complex carbohydrate combined with training have resulted in positive changes in lipoprotein components, i.e., increased HDL-Chol (Squires et al., 1979; Hicks et al., 1980). Squires

et al. were not able to demonstrate a significant difference between the training group and a group that was given dietary recommendations.

D. Diet and training upon LCAT activity

In the present study, the role of LCAT is regarded as the key enzyme related to the serum cholesterol metabolism. This enzyme converts lecithin with an unesterified cholesterol(UC) of HDL-Chol to a cholesteryl ester (CE). Also, it promotes the non-enzymatic transfer of lecithin and UC from sources such as chylomicron remnants and plasma membranes to HDL-Chol and LDL-Chol (Leiss et al., 1978; Glomset, 1979; Hoplins and Barter, 1980; Barter and Jones, 1980). This enzyme is the main factor responsible for the presence of CE in the blood within the lipoproteins, and the CE from LDL-Chol may be absorbed by arterial endothelial and smooth muscle cells and therefore contribute to atherosclerotic plaques. Also LCAT activity has an indirect effect of physiological importance. This enzyme may diminish the surface stability of VLDL-Chol and permit LPL penetration (Cramp and Trickner, 1978). In addition the UC, but not the CE, inhibits the LPL. Thus, LCAT seems to play an integrative role in TG clearance (Norby and Norum, 1978; Wallentin et al., 1978).

Dobiasova and Vondra (1978) studied lipid concentrations and expressed these as ratios of LCAT activity and TG, CE and UC. These calculated ratios clearly

show that when LCAT is low, TG are low, and when TG are high, as in obese subjects, LCAT is also high. Unfortunately, lipoprotein levels were not reported and the LCAT of this study is a fasting one, therefore the low LCAT and TG level in normal sujects may stem from their higher integrative role in LCAT activity in post-alimentary lipemia with chylomicron and VLDL-Chol and HDL-Chol. This last assumption is partly supported by the study of Rose and Juliano (1977), who showed a significant increase in LCAT activity after a high-fat test meal along with TG (r=.93, p 0.01). It seems, therefore, that normal individuals may show a lower TG level under fasting conditions mainly due to higher LCAT activity after lipid absorption. The higher LCAT activity is due to HDL-Chol concentration which is known to contain apo AI which stimulates LCAT activity. While the LCAT/CE ratio seems to be a constant, regardless of the group (Table 2), the LCAT/UC ratio tends to indicate that LCAT activity rate is dependant on the substrate level (cholesterol), i.e, low UC would be related to high LCAT activity. This contradicts Lacko et al. (1977), who showed that the correlation of LCAT levels with UC in males is r=0.78 (p 0.005). The methodology does not seem to be the reason for the discrepancy, since in both studies they used the procedure of Glomset (1968).The best explanation may be that, in the Dobiasova and Vondra study, subgroups were used (obese, hyperlipidemic, etc.) while with the Lacko et al. correlation, only normolipemic subjects were used.

Nevertheless, further study seems necessary to explain such results and to assess if a relationship exists between LCAT and other lipoprotein subfractions. Leiss et al. (1978) tends to confirm Lacko's et al. results, but they used a different technique to determine the LCAT level.

Presently, the literature contains evidence that LCAT activity may be related to CVD by the enzyme mobilization of cholesterol from atherosclerotic lesions. Unfortunately, the results are contradictary. Bernstein and Bernstein (1978) found no significant results between LCAT activity and the severity of the coronary diseased vessels. But Sodhi et al. (1980) showed that the activity of the enzyme was highest in patients with normal coronary arteries, and a stepwise decrease existed in the activity of the enzyme as the extent of the coronary artery disease increased from one to three vessels. In addition, only this last study reported lipoprotein levels while significantly lower HDL-Chol was found in triple vessel coronary artery disease compared with normal vessels. This may be due to higher apo AI in normal subjects.

Larking and Sutherland (1977) in their study found that rats fed with safflower oil compared with animals fed with butter, beef fat or normal diet, had lower UC and TG while the total cholesterol remained the same. The low LCAT activity with the experimental group is explained by the low levels of UC and TG as supported by Rose and Juliano (1976), Lacko et al. (1978), Pinon et al, (1980) and Miller et al

TABLE II.2

Lipid Concentration and Basal LCAT Activity
(modified from Dobiasova and Vondra, 1978)

Males Groups (nb)	LCAT	TG	LCAT/TG	UC	LCAT/UC	CE	LCAT/CE
normal 65	94	84	1.11	51	1.84	162	0.58
obesity 18	115	156	0.74	62	1.86	188	0.61
diabetes 29	102	239	0.45	78	1.31	201	0.020
HLP II 12	104	250	0.41	101	1.03	279	0.51
IM 37	100	239	0.42	75	1.34	206	0.49

LCAT: micro mole/li.hr, CE and UC: micromole/li, HPL II= hyperlipoproteinemia II, IM= ischemic myocardium.

(1975). Miller et al. (1975) found that LCAT activity decreased in men when the diet was rich in PUFA in men.

This seems to contradict the general theory based on the fact that LCAT has a higher specificity for esterification when the fatty acid in position two of lecithin is unsaturated (Sgoutas, 1972), and therefore, a PUFA diet should cause an increase in LCAT activity. A feasible explanation is that the LCAT activity may increase in the first stage of a PUFA diet. Furthermore, after an extended period on the diet, the enzyme activity decreases due to the lack of available substrate UC (Larking and Sutheland, 1977) and the product inhibitors CE may control the esterification rate.

At the present time, little is known of the effect of diet on cholesterol esterification in the lipoprotein

subfraction. A decrease in caloric intake, as well as a decrease in lipids, and the nature of the lipids, may vary the cholesterol composition of diferent lipoproteins, which could explain Shekelle's (1981) results more clearly. Thus, diet may influence total cholesterol mainly by a decrease in UC while CE remains the same or higher. In humans HDL-Chol increases from an appropriate diet and is negatively associated with VLDL-Chol and LDL-Chol (Hulley et al., 1979), while caloric restriction seems to decrease HDL-Chol and increase LDL-Chol as a consequence of the low turnover of exogenous and endogenous triglyceride-rich lipoproteins (Taskinen and Nikkila, 1979). However, this latter experiment lasted a few days, while the preceding was epidemiological. Nevertheless, population studies showed that lipoprotein levels are influenced by diet (Connor, 1979). A diet influencing the level of UC and CE in humans is not known, but determining these changes would add new information regarding the effect of diet on lipoproteins.

Some studies are now considering the cholesterol esterification (Yashiro et al., 1980) and are finding significant changes from training (Table 3).

First, it should be noted that mice lipoprotein levels, ratio, and metabolism differ from humans. But many parallels can be made, tempting one to suspect interesting changes using physical training on human subjects. Results in Table 3 show a remarkable increase in HDL-Chol/LDL-Chol ratio in the exercising group, a ratio that is known to be negatively

TABLE II.3

Effect of Voluntary Exercise on Mice Cholesterol and LCAT (modified from Yashiro, 1979, n= 36)

	TC	UC	CE	LCAT	HDL	LDL	HDL/LDL
NE	157.8	27.0	130.8	8.63	115.2	31.7	3.64
E	127.3	19.3	108.7	12.33	105.3	19.5	5.40

Levels in mg %.
NE= non exercise group. E=exercise group.

related to CVD. This is concomitantly related to an increase in LCAT activity. Meanwhile, changes in cholesterol levels are especially interesting when comparing CE/UC, which is 4.84 in the control group and 5.63 in the exercising group, showing an increased ratio with higher LCAT activity.

These results are in accordance with those of Simko and Kelley (1979) who had an increased LCAT activity. Lower biliary cholesterol level was explained as a result of regular exercise, which promoted the transport of peripheral cholesterol to the liver during the period of activity. This last assumption is interesting since training is known to increase HDL-Chol/LDL-Chol ratio which is considered antisclerotic.

Unfortunately, studies with human subjects attempting to determine the effect of training on cholesterol esterification does not exist. Evidence from animal studies suggest a possible role of LCAT on changes of cholesterol levels and lipoproteins. But a single relationship from LCAT

with another blood component may not be found, since the enzyme activity may depend on a combination of substrate availability, product removal and lipoproteins (Thanabalasingham et al., 1980). Only one study has shown increased LCAT activity with humans from training (Lopez, 1974).The enzymatic activity was determined in only five subjects who were medical students with a low lipid profile.

E. Blood glucose and physical fitness

Even though this study is concerned with normal adults, it is still pertinent to consider the side effects of high blood glucose and special cases, such as diabetes mellitus. Middle-aged men who are normoglycemic, but have a pathological oral glucose tolerance test (OGTT) have an increased risk of developing diabetes (Jarret and Keen, 1976), and CVD is more common among these men (Keen et al., 1965; Garcia et al., 1973; Ostrander and Lamphiear, 1976). In addition, intermittent claudication is generally more common in diabetes (Garcia et al., 1973). Myocardial infarctions from autopsies revealed a two-fold incidence from diabetes (Michell and Schawrtz, 1965). Even mild impairment of OGTT is associated with an increased frequency of atherosclerotic phenomena (Keen and Jarret, 1975), or the risk of CVD increases parallel to the response of OGTT (Amsterdam, 1977). Also, it should be noted that abnormal OGTT is associated with other CVD risk factors, i.e., obesity and hyperlipidemia. Thus, glucose intolerance should

be considered as a significant CVD risk factor because of its association with other factors.

Basal plasma insulin and plasma glucose are significantly reduced following periods of physical training in both obese (Le Blanc et al., 1978; Sterky, 1971) and normal subjects (Cooper et al., 1976; Davidson et al., 1966; Rennie and Johnson, 1974; Le Blanc et al., 1977; Ruderman et al., 1979), partly because exercise training produces an enhanced sensitivity to insulin (Le Blanc et al., 1977).

Le Blanc et al., (1979) studied the effect of training among 18 subjects, aged 18-30 years. They observed a significantly reduced insulin response to the OGTT in the better trained subjects, and fasting blood glucose was lower in the best-trained subjects. Ruderman et al., (1979) studied the effect of a training program over a period of 3-6 months. The mean age of the six subjects was 52 and they showed a high fasting blood glucose with deficient insulin secretion but none were under medication. The subjects trained at home 30min/day, five times/week with OGTT being given 6 days before training, 6 days after training and 14 days after training. Training improved OGTT, but this improvement was not maintained in the absence of exercise, as another 8 days of inactivity led to a significant decrease in glucose disappearance even though the improvement in VO2 max was maintained. Saltin et al., (1979) studied normoglycemic subjects who had a pathological OGTT (or chemical diabetes) with a mean age of 48 and were not

more over-weight than age-matched controls. Group A trained two times/week and received appropriate diet information at the beginning (n=25; observations made at 0, 6 and 12 months). Group B trained as group A but without dietary information (n=11 ; observations made at 0 and 3 months). Group C received only dietary information. The results showed that men with chemical diabetes had a 20% lower VO2 max. VO2 max increased with only dietary advice but was insignificant for several men in the group. OGTT improved for all three groups but was normalized only in group A. Insulin levels during OGTT were reduced for groups A and B, however, the reduction in plasma insulin concentration was insignificant in group C after three months. Finally, Cooper et al., (1976) on a large scale epidemiological study of 2,998 men with an average age of 44.6 years, observed a significantly higher glucose level from the very poor (111.0 mg%) to poor (107.3 mg%), compared to excellently fit (103.4 mg%) subjects.

The mechanism normally related to that change in OGTT and blood glucose can be explained. Normally by their training highly fit parsons are used to high catecholamine secretion which facilitates repeated enhanced catecholamine secretion by severe exercise which gradually diminishes the capacity of the pancreas to secrete insulin (Von Euler, 1974; Le Blanc, 1979). This latest explanation might not describe the results of Saltin (1979), where the subjects were submitted to a mild training program. Saltin suggested

that a small quantity of muscle glycogen remaining after prolonged exercise will increase the proportion of glucose passing the liver and permit more glucose to be taken up by peripheral tissues. Finally, Ruderman (1979) suggested that physical training, of a previously inactive patient with mature-onset diabetes, enhances his ability to dispose of an intravenous glucose load due to an enhanced sensitivity to endogenous insulin or a diminished anti-insulin factor.

F. Serum uric acid and physical fitness

Among the most important factors associated with uremia are hypertension, coronary artery disease, hypertriglyceridemia, high cholesterol, alcoholism, diabetes mellitus, hyperlipemia, obesity, renal failure, primary gout, and anxiety (Brekenridge, 1966; Bosco et al., 1970; Klein et al., 1973; Montoye et al., 1975; Tweeddale and Fodor, 1979). It is improper to label high SUA as a direct risk factor; however it is best termed as an indicator, because the mechanism by which a high SUA level is associated with future atherosclerosis or hypertension remains to be shown (Tweeddale and Fodor, 1979; Fessel, 1980). Therefore, any conclusions or explanations for associations found between CVD risk factors and SUA levels must be made with caution (Klein et al., 1973).

Many studies have shown the important correlation between SUA and hypertension. Fessel et al., (1973) studied the SUA in two groups of adults over a period of fifty

months. In group A (hyperucemic - 124 subjects) hypertension developed in 18 subjects, whereas in group B (normal - 224 subjects) hypertension developed in only 3 subjects, It was concluded from statistical analysis that hyperuricemic subjects had a 10.8 times greater risk than control subjects. Later (1980), the same author investigated the hypothesis that body weight is not the factor underlying the relation between hyperuricemia and CVD. Among 111 subjects with hyperuricemia, studied for 108 months, 25 of the group developed hypertension and their mean weight was not significantly higher. In this same study, Fessel outlined another study. From 1,356 persons aged 60 to 69 years who had their SUA recorded in 1967, subsequent deaths from CHD showed a stepwise increase when deaths were arranged according to the SUA levels but not when they were arranged according to body weight. It was concluded that hyperuricemia predicts future CVD independent of body weight. This last statement is not supported by Klein et al., (1973) who concluded that the prevalence of hypertension was greater in hyperuricemics as compared to normouricemics in all race-sex groups, but increased prevalence of CVD in hyperuricemics was secondary to increased body size. Although the experimental design is different between the two studies, one (Fessel 1980) studied the relation of SUA with hypertension and death, while the other (Klein et al., 1973) correlated SUA with electrocardiogram abnormalities and hypertension.

SUA is known to increase (up to 2.7 mg%) (Żachau-Christiansen, 1959) during exercise and the principle mechanism related to this change is mainly the decrease in renal function during that period of time. What is more interesting is the chronic effect of exercise on SUA.

Bosco et al., (1970) studied the effects of eight weeks of chronic training on SUA in thirty males, aged 18 to 29 years. Ten were extremely active (group A), ten were moderately active (group B) and ten were relatively sedentary (group C, control). SUA concentration and the Harvard step were measured at the beginning, periodically and after a four week "deconditioning" period. The pre-experimental correlation coefficient between SUA and the fitness index were low, however, chronic physical exercise lowered SUA from 0.3 to 3.2 mg% in 80 percent of the subjects from groups A and B, particularly in those persons with high values (7.0 to 8.5 mg%). These high SUA levels were previously reported (Montoye, 1967; Greenleaf et al., 1969) and they were related to high levels of achievements and leadership (Brooks an Mueller, 1966).

Epidemiological studies did not give a strong correlation between fitness and SUA. Montoye et al., (1978) tested 793 males and 80 females, ages 10-69, on a treadmill for VO2 max, which he related to the sum of four skinfolds, SUA and a one hour glucose tolerance test. Body fatness was positively correlated with SUA and blood glucose and

negatively correlated with VO2 max. Age was negatively correlated with VO2 max and positively correlated with blood glucose. After removing the effects of age, weight and fatness, the correlations of VO2 max and SUA were low, but some results of this study seem doubtful since the subjects were not necessarily fasted for the SUA test and this could be important enough to give a wrong estimate of SUA. Cooper et al., (1976) obtained a slight relationship between SUA and VO2 max and his finding might be taken more seriously since he obtained a P of .05 when he compared the SUA levels of the very poor, poor, and fair groups to the group with excellent physical fitness (Table 4). The factors that could have contributed to the lowering of SUA are not well understood. It is known that metabolic changes resulting from acute exertion would tend to inhibit renal urate excretion. Ketone bodies, beta-hydroxybuterate and acetoacetate retard urate excretion and lactacidemia impairs uric acid excretion (Cannon et al., 1966; Bosco et al., 1970). Therefore, this situation should increase SUA. Bosco et al., (1970) explained the chronic decrease in SUA from the possible increase in the plasme volume (up to 20%) which would give a larger pool for SUA but he rejected the avenue of the loss of uric acid from body sweat. Conversely, it is known that acclimatization to work shows a decrease in electrolytes (Consolazio et al., 1962; Smiles and Robinson, 1971) and concentrations in sweat are normally related to a decrease in plasma concentration.

TABLE II.4

Serum Uric Acid Versus Levels of Physical Fitness, uric acid in mg%
(2,990 men, mean age 44.6) (Cooper et al., 1976)

Level of Fitness	Uric Acid
Very poor	6.7
Poor	6.8
Fair	6.7
Good	6.5
Excellent	6.4

From the above studies, it is likely that a physical training program leads to a decrease in plasma SUA. This decrease should not be viewed as a metabolic change since some exercise metabolites impair renal uric acid excretion. The main factor, however, should be the increase in plasma volume. Secondly, the uric acid in perspiration should be considered since training programs add the effect of sweat loss. Overall a significant amount of uric acid is lost from this route, particularly in the first phase of the training program.

G. Blood pressure and physical fitness

Epidemiological studies have clearly shown even mild hypertension leads to a lower life expectancy (Waaler, 1977), not to mention that both systolic and diastolic pressure increase with age for both sexes (Hamilton et al., 1954; Humerfelt, 1963). Primary hypertension is of

undetermined cause, while secondary hypertension is related to renal disease, endocrine disorders, neurologic disorders, etc. (Sokolow and McIlroy, 1979). Hypertension is known to be highly related to CVD (Robers, 1975) probably due to an increased rate of atherosclerotic deposition (Cousineau,1980).

Populations consuming very little sodium chloride have low levels of blood pressure (Prior et al., 1968; Shaper, 1972; Kesteloot et al., 1978). But proper nutrition, frequent exercise, and lower stress are also considered important as preventive measures against hypertension (Tibbin and Eriksson, 1980).

Significantly lower systolic blood pressures (122.9 mmHg) were found in highly fit individuals compared with (127.6mmHg) higher pressures in low fit subjects (Cooper, 1977). A significant reduction in blood pressure caused by physical training has been noticed in sedentary 40 year old men (Kilbom et al., 1969) and older normal subjects (De Vries, 1970). Subjects considered to be borderline hypertensives also show a significant decrease (Choquette and Ferguson, 1973) with training.

The mechanism believed to be responsible for lowered blood pressure from training is a reduced activation of the sympathetic system during maximal work or at the same relative work load (Cousineau, 1980).The reduced circulating catecholamines have been related to the reduced heart rate and systolic blood pressure (Cousineau et al., 1977) during

different levels of work after a period of training, but no significant changes in resting catecholamines in the pre and post test were found. It seems therefore, that mechanisms other than catecholamine levels may play a role in reducing blood pressure in trained individuals.

H. Effect of training on body weight and cardiac response

The effect of physical activity on body weight and cardiac response to work is well documented and accepted (Astrand and Rodahl, 1970; Bouchard et al., 1974; Edington and Edgerton, 1976). Actually, the norms to predict cardiovascular fitness are VO2 max values which are significantly related to good physical fitness, but predicting VO2 max from submaximal work has some problems. In addition, VO2 max is not necessarily an endurance type of measure when considering new literature dealing with the aerobic and anaerobic threshold (Skinner and McLellan, 1980), i.e., some individuals may be able to sustain for a long period of time a high level work (80% VO2 max), while others may have difficulty in sustaining a lower level of work (60% VO2 max) and these people are normally less fit. While the majority of authors use submaximal goals (Cureton, 1965; Astrand, 1970; Bouchard et al., 1979) to define physical fitness, i.e., an efficient oxygen transport system for daily work and additional strenuous activities, Shephard (1976, 1978) tends to relate cardiorespiratory fitness to VO2 max. No authors mentioned that cardiovascular fitness is

VO2 max. For these reasons, this study will use the heart rate response to the Standardized Test of Fitness (STF) step test (Fitness and Amateur Sport Canada, 1981) to assess cardiovascular fitness.

I. Menstrual cycle, working capacity and blood composition

Women, partially due to their hormonal secretion (Nikkila, 1978, 1979), have a significantly lower level of cholesterol, LDL-Chol, VLDL-Chol, T.G., blood glucose, SUA (LRC Data Book 1978), while the level of HDL-Chol and HLD-Chol/LDL-Chol are higher compared to men. These beneficial differences are considered as being very important variables for reduced susceptibility of premenopausal women to atherosclerosis (Kannel et al., 1976).

Plasma lipids are influenced during the menstrual cycle. HDL-Chol, phospholipids and triglycerides are fairly stable throughout the period (Punnonen, 1978; Kim and Kalkhoff, 1979; Basdevant, 1981). The LDL-Chol and LDL-ApoB are relatively lower during the luteal phase (Kim and Kalkhoff, 1979). Also, these authors observed a significant decrease in total cholesterol during the luteal phase while Basevant et al. (1981) showed a decrease, although it was insignificant. Therefore, the period of the menstrual cycle of women should be considered when plasma lipids are measured: LDL-Chol may vary from 110 to 90 mg% and HDL-Chol/LDL-Chol ratios from 0.6 to 0.75 when compared

between the menstrual phase and luteal phase (Kim and Kalkhoff, 1979).

While it is well documented that physical activity influences the length of menstrual cycle (Shangold et al., 1979) and the age at menarche (Malina et al., 1978), the influence of different phases of the menstrual cycle upon working capacity tends to indicate a reduced working capacity during menstruation and the highest values during the period of ovulation (Doskin et al., 1980). Therefore, when women's physical work capacity or serum composition are tested, it seems reasonable to consider the period of menstrual cycle as a relative value in pretest and postest design. Although, for total values it seems less important.

J. Conclusion

Even though physical fitness is very important for an individual's health it should not viewed as the panacea for any disease. In this review an attempt was made to use studies dealing with healthy human subjects, therefore the effect of physical training in obese, diabetic or cardiac rehabilitation groups should be considered specific to these populations. In order to delimit the study from a practical point of view, cigarette smoking, stress and some other factors are not considered.

Almost all the studies in this review have dealt with young subjects involved in a fairly comprehensive training program. The sample of this study consists of unfit adults

observed under a program which is widely used. The volunteers will be observed on many variables normally not evaluated in the previous literature. Another factor that is not well covered in the literature is the changes in UC and CE and how their distribution may change among lipoproteins.

III. METHODOLOGY

A. Subjects

Seventeen male and twenty four female sedentary volunteers between the ages of 30 and 58 participated in the study. A person was considered sedentary if he did not train on a regular basis and had led a relatively sedentary life for the previous two years. The subject had to show evidence of good health to participate in a physical fitness program. Recruitement of the subjects was made with the help of the YMCA. The participating subject had to pay seventy-five ($75) dollars to be enrolled in the fitness class. Prior to the study it was advertised three times in the newspaper and it was publicized as a fitness study. Control subjects (FEM.C n=6, MAL.C n=6) were recruited by asking volunteers to find an age, sex and activity level matched individual.

Before the beginning of the experiment subjects were asked to sign an informed consent form and complete the "Par Q" questionnaire (Appendix A).

B. Procedures

Subjects were tested in the morning after a 12 hour fast and a 72 hour alcohol restriction. They were asked to refrain from exercise for 12 hours before the test. Women were asked to record the date of their last menstruation in the pretest and to return for the postest in the same period, while for the men it was the same day of the week on

both occasions.

The following dependent variables were measured before the training program and at the end (12 to 15 weeks):

Weight (Kg)

Skinfolds for body fat (%) prediction

Resting systolic blood pressure (mmHg)

Resting diastolic blood pressure (mmHg)

Post exercise heart rate from the STF step test in beats/min

Physical activity (min/2wk)

Daily caloric intake (Cal/day)

Daily fat intake (gr/day)

Daily cholesterol intake (mg/day)

Daily saturated fat intake (gr/day)

Daily PUFA intake (gr/day)

Total serum cholesterol (mg/100ml)

Total serum free cholesterol (mg/100ml)

Serum triglyceride (mg/100ml)

Blood glucose (mg/100ml)

Serum uric acid (mg/100ml)

Serum HDL-Chol (mg/100ml)

Serum LDL-Chol (mg/100ml)

Serum VLDL-Chol (mg/100ml)

Free cholesterol in HDL-Chol

Free cholesterol in LDL-Chol

Free cholesterol in VLDL-Chol

LCAT IRE

LCAT activity (micromol/li.hr)

Body composition, diet and activity record

Body weight was recorded in kilograms. Body fat was measured using Harpenden skinfold calipers at the following sites: triceps, subscapular, biceps, and suprailic (Fitness and Amateur Sport Canada, 1981). Percent body fat was estimated from the sum of the four skinfolds by the use of a table (Durnin and Womersley; 1974).

Diet characteristics were monitored for three successive days (Sunday, Monday and Tuesday) with the aid of a questionnaire (Appendix A) asking them to list all the food items and quantity they ate during those days. Weekly activity was recorded for the two weeks prior to the pre test and the postest (Appendix A).

Resting blood pressure

Blood pressure was the first measure made. It was recorded with the subject seated for a minimum of three minutes, with subsequent measurements of systolic and diastolic blood pressure taken on the left arm.

Standardized test of fitness

The standard procedure was followed in administering the STF step test (Fitness and Amateur Sport Canada, 1981). The heart rate response at the last level of work was recorded as the post exercise heart rate. The heart rate was

recorded at that same level of work in the postest even though the subject may have been subjected to a higher level of work on the second occasion.

Blood analysis

The blood was taken by a trained technician, from the antecubital vein, after the blood pressure recording. About ten milliliters of blood were drawn in a silicone coated vacutainer. The plasma of this sample was used for lipoprotein determination (HDL-Chol, LDL-Chol and VLDL-Chol). About three milliliters of blood were drawn in a sodium heparinized vacutainer and immediately put in crushed ice. The plasma of this sample was used for the determination of LCAT activity, triglyceride, serum glucose and serum uric acid. The blood sample was spun in a centrifuge at 3,000 g's for 10 minutes. The serum was transfered to a small test tube (100x12mm) and frozen (at -60 degrees Celsius) for further analysis. The samples were analyzed within two months.

Serum glucose was determined by a colorimetric method using Sigma (1977a) kit 635. SUA was determined by an ultraviolet absorption method using the Sigma (1976) kit 292-UV. Serum TG were determined by the colorimetric method by using the Sigma (1977b) kit 405. Cholesterol and esterified cholesterol was determined by the enzymatic method of Roschlau, Bernt and Gruber (1974). HDL-Chol was isolated by the heparin and manganese chloride precipitation

method. The LDL-Chol and VLDL-Chol was isolated by fractionation flotation by the method of Hatch and Lee (1968). LCAT activity was determined by the method of Dieplinger and Kostner(1980). The procedures for serum analysis are described in Appendix C.

C. Experimental design and training program

All volunteers were invited at the same time to sign up in group A or B. In order to avoid undesired grouping, the subjects had to put their name on a sheet where space was available to sign up limited by age (30 to 35, 36 to 41, 42 and over) and sex. For example, it was known from the pretest that only eight places for women between 30 to 35 were available, therefore only four spaces were made avaible in each group. The actual difference in the number of subjects per group is due to a slightly higher registration in group B and a higher drop-out rate in group A. Group A (FEM.A n=8, MAL.A n=4) trained on Tuesday from 8 to 9 pm and on Thursday from 7 to 8 pm. Group B (FEM.B n=10, MAL.B n=7) trained on Tuesday from 7 to 8 pm and on Thursday from 8 to 9 pm. The program started on September 15 and was completed on December 18, 1981. Each subject kept a weekly record of his activity in the YMCA attendance book. The subjects trained three times a week, twice with a YMCA instructor, while the third session was undertaken without supervision.

All trained subjects (group A and B) received fitness information from their instructor and from popular

pamphlets. The dietary information given to one group (B) was in the same form as for the ones in the training group (A). Subjects were asked to refrain from exchanging information with the other group. In addition, they were encouraged to follow dietary recommendations but this was not compulsory (pamphlets were distributed as described in Appendix B). During the experiment there was no specific approach to the problem of cigarette smoking or stress. Subjects were asked to monitor their training in order to exclude those who did not train properly. A typical lesson and the progression in training intensity is described in Appendix D.

D. Statistical analysis

A three way ANOVA with repeated measures was done to determine the significance of differences between and within groups for the following dependent variables; body weight, percent body fat, systolic blood pressure, diastolic blood pressure, diet characteristics, blood measures, ratios from lipoproteins, cholesterol, triglycerides and LCAT determinations. Ratios analysis of variance were done when the analysis of a single parameter showed a trend or a significance and also when there was a physiologic meaning to testing the difference between two means.

The program used for the analysis of variance was the "BMDP Biomedical Computer programs" from Dixon and Brown (1979). For the post hoc analysis the Scheffe method was

used to compare the means (Ferguson, 1971). Critical mean difference was calculated from the multiple comparison procedure of Scheffe (Ferguson, 1971): critical mean difference = square root of (F((mean square error/n1) + (mean square error/n2))), where F was the value from the table and n was the number of subjects per group.

IV. RESULTS AND DISCUSSION

The results and discussion are presented in seven sections: body composition, physical activity (min/2wk), post exercise heart rate, blood pressure, diet characteristics, serum glucose and uric acid, and finally serum lipids.

A. Body composition

Percent body fat changes are presented in Figure 1 and total body weight in Table 5. There was a significantly lower body weight and greater fat percentage for women, and this was in accordance with anthropometric charts comparing men and women. There were no significant changes in predicted percent body fat and total body weight during the study. Similar lack of significant changes were previously observed Holloszy et al., (1964). The lack of significant changes may be due to the short duration of the program, the intensity of training and/or to the unchanged food habits of the investigated subjects.

B. Physical activity

Total physical activity is shown in Figure 2, with both training groups showing a significant increase (p=.001) in their activity related to fitness improvement. As mentioned in Appendix D, the volunteers increased their physical

TABLE IV.5

Changes in Mean Body Weight, Predicted VO2 max, Serum Glucose and Uric Acid

GROUP	FEM.A	FEM.B	FEM.C	MAL.A	MAL.B	MAL.C	TOTAL
Changes in mean body weight in Kg							
Pre.	65.57	63.13	64.93	76.75	76.70	89.33	71.35
S.D.	9.41	14.95	10.28	13.30	8.10	21.12	
Post	65.00	63.17	64.85	77.25	76.30	89.40	71.23
S.D.	8.51	15.19	9.13	14.78	7.79	20.21	
Changes in mean predicted VO2 max ml/Kg.min							
Pre.	25.40	28.09	31.03	32.50	36.19	35.00	30.82
S.D.	2.66	5.64	3.90	5.38	2.73	8.18	
Post	28.89	30.45	30.70	34.75	38.77	35.78	32.80
S.D.	3.89	5.63	3.74	4.91	2.34	8.16	
Changes in mean serum glucose in mg/100ml							
Pre.	100.5	94.1	95.1	99.7	89.7	97.3	95.8
S.D.	10.8	12.1	7.3	7.0	9.8	7.6	
Post	100.2	95.1	93.0	98.1	92.8	95.8	95.8
S.D.	12.3	12.7	8.3	3.6	9.5	4.6	
Changes in mean serum uric acid in mg/100ml							
Pre.	4.853	5.135	4.465	6.237	5.500	5.433	5.196
S.D.	1.357	1.325	1.121	1.759	1.316	1.355	
Post	4.693	5.240	4.550	6.475	6.021	5.425	5.313
S.D.	1.434	1.493	1.215	0.972	0.891	1.199	

activity by classes in the program and also by changes in lifestyle (Appendix A). That increase in activity corresponded to the minimal level of recommended quantity and quality of exercise for developing fitness in healty adults (Amer. College of Sports Med., 1980), which is:

1. Three to five days per week.
2. Training level of 60 to 90% of maximum heart rate reserve.
3. A continuous aerobic activity of 15 to 60 minutes.

4. Any activity that uses large muscle groups, that can be maintained continuously, and is rhythmical and aerobic in nature.

C. Post exercise heart rate

There was a significant improvement in step test results of both male and female trained subjects ($p=.001$). However male controls also increased their fitness ($p=.05$), but that increase was lower than for the trained subjects ($p=.001$)(Figure 3). One subject in the male control had shown to be fairly unconfortable with the blood test on both occasions so although he was fairly young (30) the step test was stopped at 138 beats per minutes on the first test thereby making it easier for him on the second test which was recorded at 120 beats per minutes. Also one other male control subject, age of 51, was tested only on level one because of his bulky weight of 120kg and he showed a decrease of 12 beats per minute on the second test. Therefore, 5 beats per minute of the mean decrease of 6 beats per minute on the second test in the male control group was from these two subjects. That change may be more related in one case to psychological factors while in the other case to a low level of work in the first test.

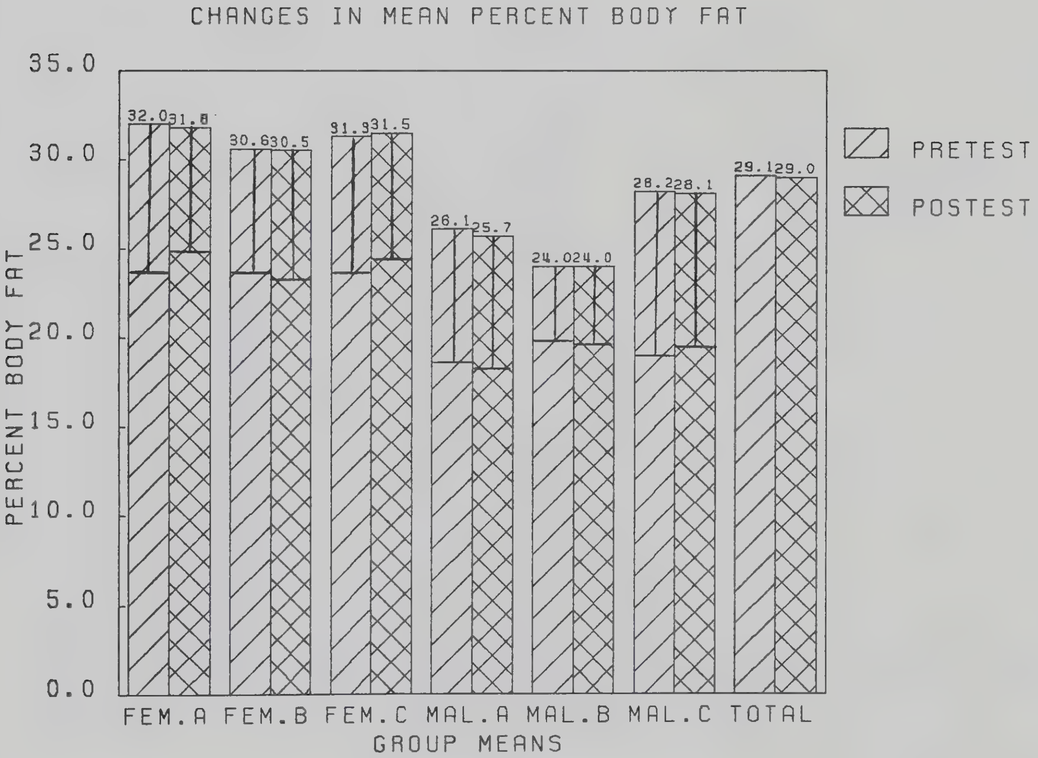
The predicted VO2 max ml/kg.min (Table 5) showed that the recruited subjects were at a minimum level of fitness according to the norms (Can. Pub. Health Assoc. 1977) and thus prone to increase their level of fitness. The analysis

of variance in treatment by time (Appendix E) of predicted VO2 max showed a similar improvement in fitness as the one in the post exercise heart rate. Similar increases of 10% in predicted VO2 max were observed for a shorter period of training (7 weeks) at more a intense level twice a week (Pedersen and Jogersen, 1978). However, the lower F value in predicted VO2 max comes from the integrated values of individual's weight and age of the Jette et al. formula (1976) and those two values did not change.

D. Resting blood pressure

There was a significant decrease in systolic blood pressure (p=.05) and a tendency (p=.07) towards a decrease in diastolic blood pressure in the training groups A and B (Figures 4 and 5). Similar changes have been previously reported (Kilbom et al., 1969; De Vries, 1970) and the reduced sympathetic activity was considered as having an important role in this change (Cousineau, 1980). Group MAL.A had a mean change of 137 to 128mmHg (p.01) in systolic blood pressure and a mean decrease from 96 to 89mmHg (p.01) in diastolic blood pressure. Choquette and Ferguson (1973) reported similar changes from bordeline hypertensive subjects. Even though the subjects have been tested in similar conditions by the same technician, it should be noted that the trained subjects (group A and B) were more accustomed to that person from the frequent contact in the YMCA's classes and therefore less psychologically stressed

FIGURE IV.1



=Standard deviation

FIGURE IV.2

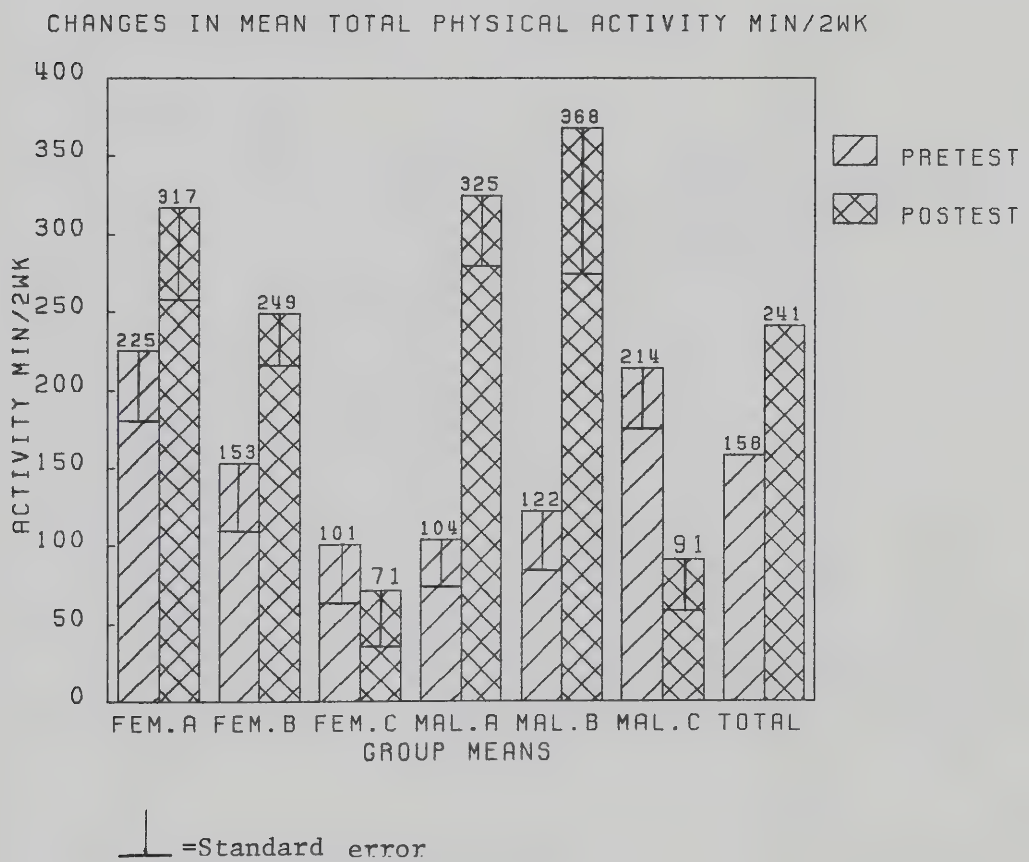
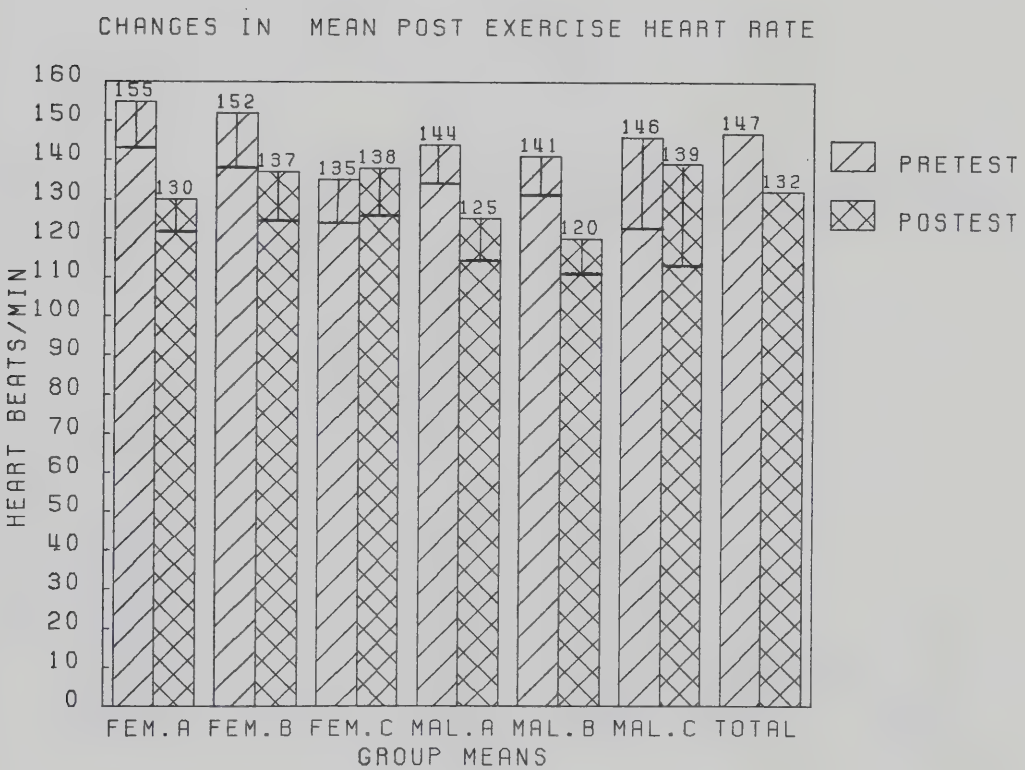

CHANGES IN MEAN TOTAL PHYSICAL ACTIVITY MIN/2WK
ACTIVITY MIN/2WK
0
50
100
150
200
250
300
350
400
PRETEST
POSTEST
225
317
153
249
101
71
104
325
122
368
214
91
158
241
FEM.A FEM.B FEM.C MAL.A MAL.B MAL.C TOTAL
GROUP MEANS
=Standard error

FIGURE IV.3



=Standard deviation

FIGURE IV.4

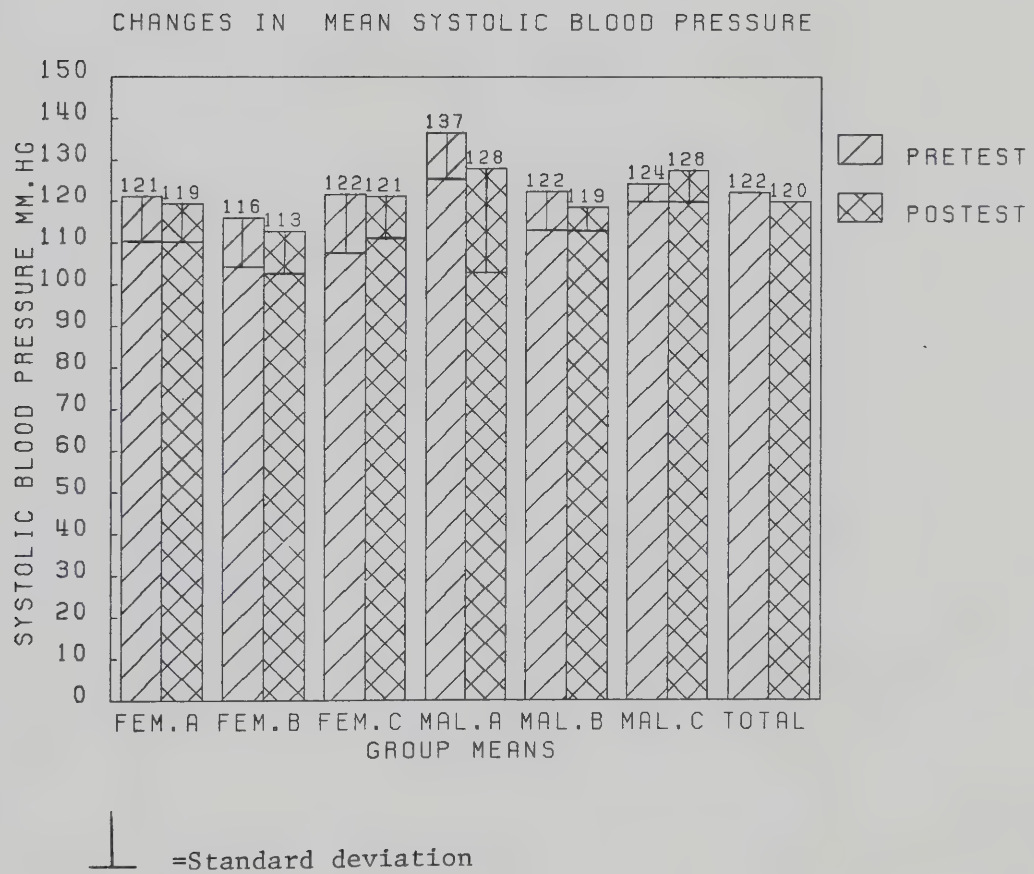
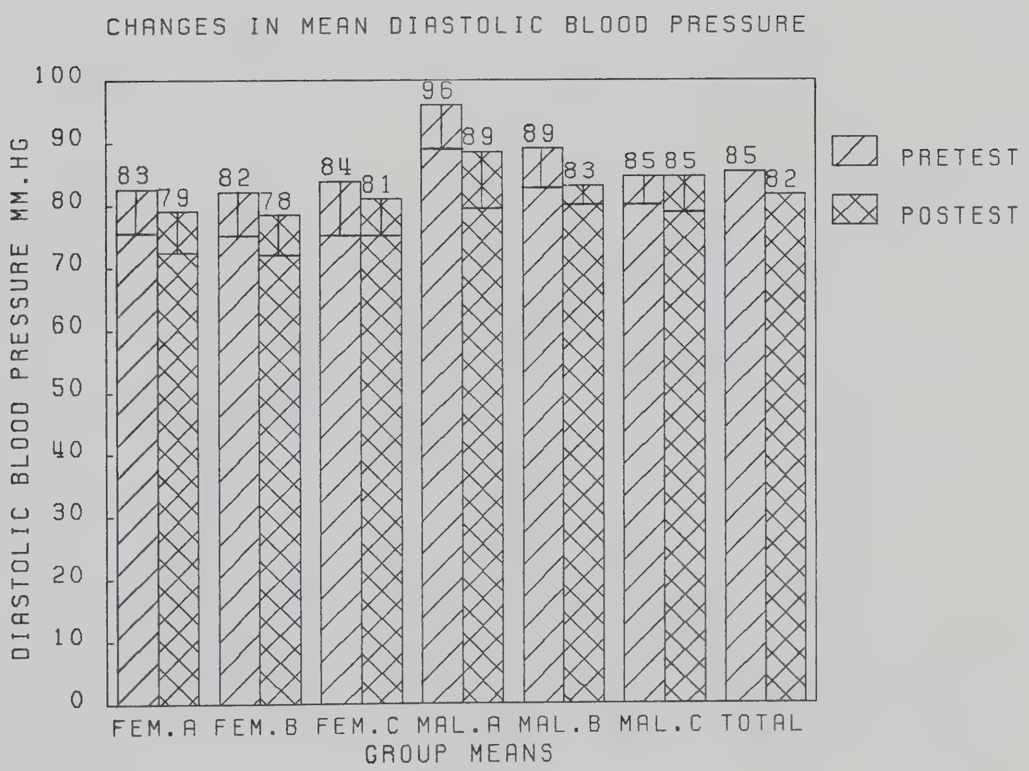
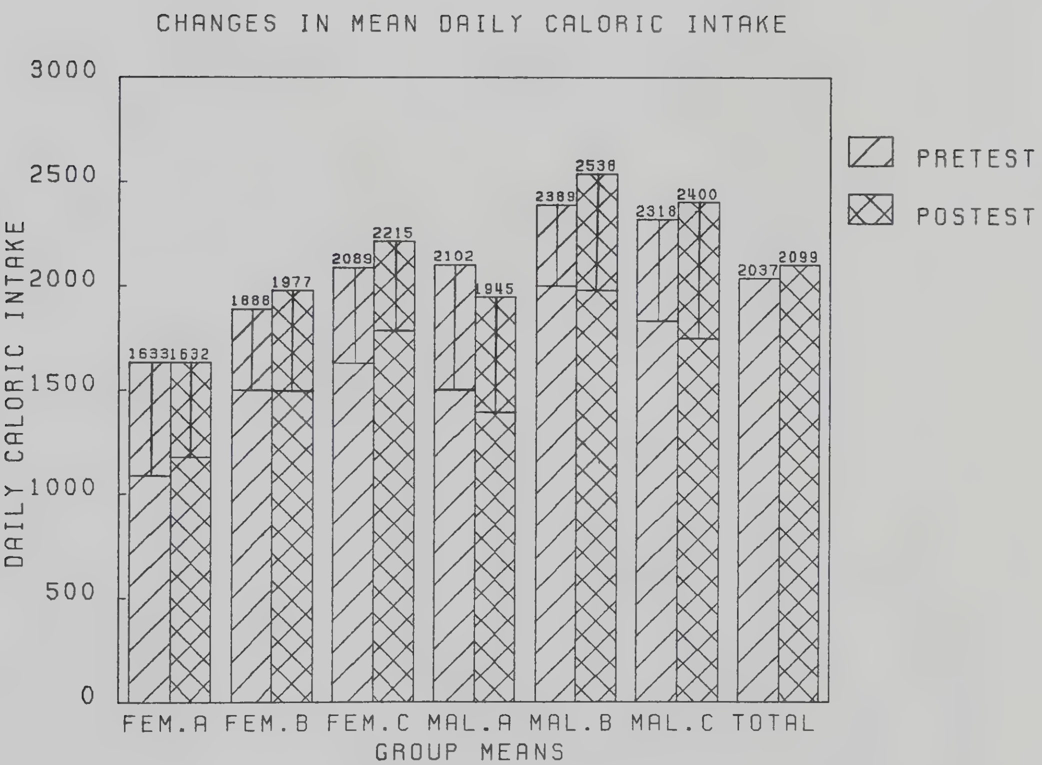

CHANGES IN MEAN SYSTOLIC BLOOD PRESSURE
SYSTOLIC BLOOD PRESSURE MM.HG
150
140
130
120
110
100
90
80
70
60
50
40
30
20
10
0
121
119
116
113
122
121
137
128
122
119
124
128
122
120
PRETEST
POSTEST
FEM.A
FEM.B
FEM.C
MAL.A
MAL.B
MAL.C
TOTAL
GROUP MEANS
=Standard deviation

FIGURE IV.5



=Standard deviation

FIGURE IV.6



=Standard deviation

in the second blood pressure test. Nevertheless, within the experimental limitation, the actual fitness program was shown to be effective in reducing resting blood pressure.

E. Diet characteristics

One of the biggest concerns among women subjects, and to a lesser extend also for men, was their weight. They were not only motivated to change regarding their total activity, but also their food habits. Their percent body fat showed that they were above average (Can. Pub. Health Assoc. 1977) and many of them were anxious regarding their weight gain during the previous year. Therefore, it was suspected that the grouping for training (group A) and training plus diet information (group B) would be successful and that group B would positively modify their food style from the information given (Appendix B).

Total caloric intake measures indicated that the experimental population was lower than the normal Albertan diet intake (Nutrition Canada, The Alberta Survey Report, 1975). This may be due to errors in the analysis of dietary recall, although it did not seem to be the case since the diet composition in the tested population was closely related to the Alberta Survey. So, the lower level may be due more to the fact that the tested population was sedentary.

The analysis of variance showed no significant change during treatment (Appendix E) in total caloric intake

(Figure 6), total cholesterol intake, total fat intake, total PUFA intake and total saturated fat intake (see Table 6 for means and standard deviations). Therefore, even though group B received more information related to diet and cardiovascular disease, it would appear that these pamphlets alone were not sufficient to influence the diet composition. Arntenezius et al. (1978) obtained more positive changes but their study lasted a year and the subjects had a consultant which may have increased the effect. Time may also have been an interrelated factor in the study of Maccoby et al., (1977). So, changing the foodstyle of adults is more than being informative, as in the present study, and results may be shown to be more positive over a longer period.

The present results show that adults are reluctant to change their foodstyle, even if they show a degree of willingness to do so. The nutritionally informed group B possibly became more concerned about the importance of eating, thus explaining their increased food intake. This is shown by the increased means in caloric and fat intake of group B while group A had no similar change.

Therefore the popular pamphlets as they are presently distributed or as they are routinely given in many fitness classes, should be discussed and explained by the instructor if the positive effect of dietary changes in fitness classes is an important goal. The instructor must actively approach the problem.

TABLE IV.6

Diet Characteristics

GROUP	FEM.A	FEM.B	FEM.C	MAL.A	MAL.B	MAL.C	TOTAL
Total fat intake gr/day							
Pre.	64.50	82.25	89.10	91.75	88.56	94.75	83.62
S.D.	18.00	24.19	28.65	17.97	23.00	44.12	
Post	59.23	91.75	97.60	82.80	112.2	88.00	88.34
S.D.	26.56	32.10	23.39	14.92	37.77	38.19	
Total saturated fat intake gr/day							
Pre.	22.11	18.85	37.02	33.22	29.33	33.18	27.43
S.D.	9.81	8.08	15.44	16.46	12.52	12.84	
Post	21.06	24.67	36.83	27.03	38.28	31.22	29.26
S.D.	8.61	17.95	11.44	15.67	20.36	8.27	
Total PUFA intake gr/day							
Pre.	5.275	6.270	7.050	4.925	6.586	5.883	6.056
S.D.	1.846	1.591	4.408	3.377	1.848	4.577	
Post	5.087	6.470	7.350	4.050	10.78	6.316	6.807
S.D.	2.555	2.682	3.270	2.318	4.300	3.662	
Total cholesterol intake mg/day							
Pre.	343.6	356.3	325.0	324.5	393.1	502.3	373.8
S.D.	102.7	193.7	153.4	163.3	115.0	275.2	
Post	248.1	581.6	291.5	210.5	404.0	561.0	404.5
S.D.	129.7	690.4	134.1	69.3	166.8	378.4	

F. Serum glucose and uric acid

There were no significant changes in serum glucose and uric acid, either from treatment or time (see Appendix E for analysis of variance and Table 5 for means and standard deviations). Thus, as previously reported in the review of literature, changes in these blood levels are not always found as a result of a fitness program (Ruderman et al., 1979). In the present study, this may be due to the intensity of training, the duration of the program or the

testing procedure, i.e., fasted serum glucose instead of an OGTT as was done by LeBlanc (1979) and Saltin (1979).

G. Serum lipids

The main purpose of this study, was to investigate how the serum lipid metabolism may be modified by a fitness program. The results showed that the trained subjects improved their level of fitness, but this is not concomitantly related to the calculated lipid parameters of this study. Therefore, in the design of this study, none of the analysis of variance on cholesterol, cholesterol ester, lipoporteins, LCAT and triglyceride showed a significant change by time or treatment (Appendix E). The serum lipid values reported in this study are similar to those documented in the literature (LRC Data Book, 1978) in total values as well as differences between sexes. Although in the present study LCAT IRE was lower than those of Dobiasova and Vondra (1978) the difference was very small (6.34% versus 6.69%) and they were using younger subjects. A sex difference has been reported showing a lower mean for women than men with a high variability (Dobiasova and Vondra, 1978; Dieplinger and Kostner,1980).

A decrease in mean total serum cholesterol occured in the experimental groups (FEM.A from 195 to 192 mg%, MAL.A from 211 to 205mg%, and MAL.B from 200 to 199mg%: Figure 7) but this was not within the critical mean difference of 5.09mg% (at p.05, when n=6/gr). With an assay error of 3.4%

TABLE IV.7

Serum Lipids

GROUP	FEM.A	FEM.B	FEM.C	MAL.A	MAL.B	MAL.C	TOTAL
HDL free cholesterol mg%							
Pre.	15.71	14.42	17.99	19.00	16.96	15.63	16.25
S.D.	3.50	2.86	4.81	4.14	2.95	2.83	
Post	15.68	14.72	17.05	18.22	16.48	15.87	16.06
S.D.	3.65	2.44	5.11	3.61	2.86	2.34	
LDL free cholesterol mg%							
Pre.	32.04	34.56	28.02	38.90	36.11	33.57	33.65
S.D.	4.813	8.192	5.563	6.852	6.259	9.252	
Post	31.35	33.57	27.90	37.98	34.86	31.95	32.72
S.D.	5.677	8.879	5.554	7.640	5.086	8.200	
VLDL Free Cholesterol mg%							
Pre.	7.38	7.57	7.54	8.89	8.08	8.46	7.87
S.D.	3.77	3.29	3.05	5.14	1.82	2.50	
Post	7.29	7.58	7.04	8.75	8.23	7.73	7.69
S.D.	4.05	2.71	2.88	4.93	1.93	1.58	
LCAT activity							
Pre.	79.92	78.69	64.60	96.90	101.7	106.1	86.60
S.D.	25.50	29.33	36.42	28.00	41.42	14.63	
Post	79.20	82.54	68.30	112.1	103.2	105.2	88.15
S.D.	22.97	32.94	37.48	34.14	43.44	16.75	

such differences would have been detected. Similar types of insignificant lipid changes with an increase in VO2 max from 15 to 25%, have been previously reported: Holloszy, 1964; Goode et al., 1970; Lopez, 1974; Allison et al., 1980; Hunt and White, 1980; Ready, 1980. Some other studies reported changes in lipids with training, but they are also related to higher differences in VO2 max and changes in total body weight (Hartung and Squires, 1978; Hicks et al., 1980).

Serum free cholesterol showed a mean decrease from 59.25 to 58mg% in group A and B (Figure 8) although these changes are not close to the critical mean difference needed for a significant test. This was mainly due to the high intra-variability of the subjects, i.e., many of them decreased their total free cholesterol while some others increased which made the result insignificant. These results were different than the mean decrease of 17mg% of AlteKruse and Wilmore (1974) although training program differences and no control in dietary control may be the sources of such discrepencies.

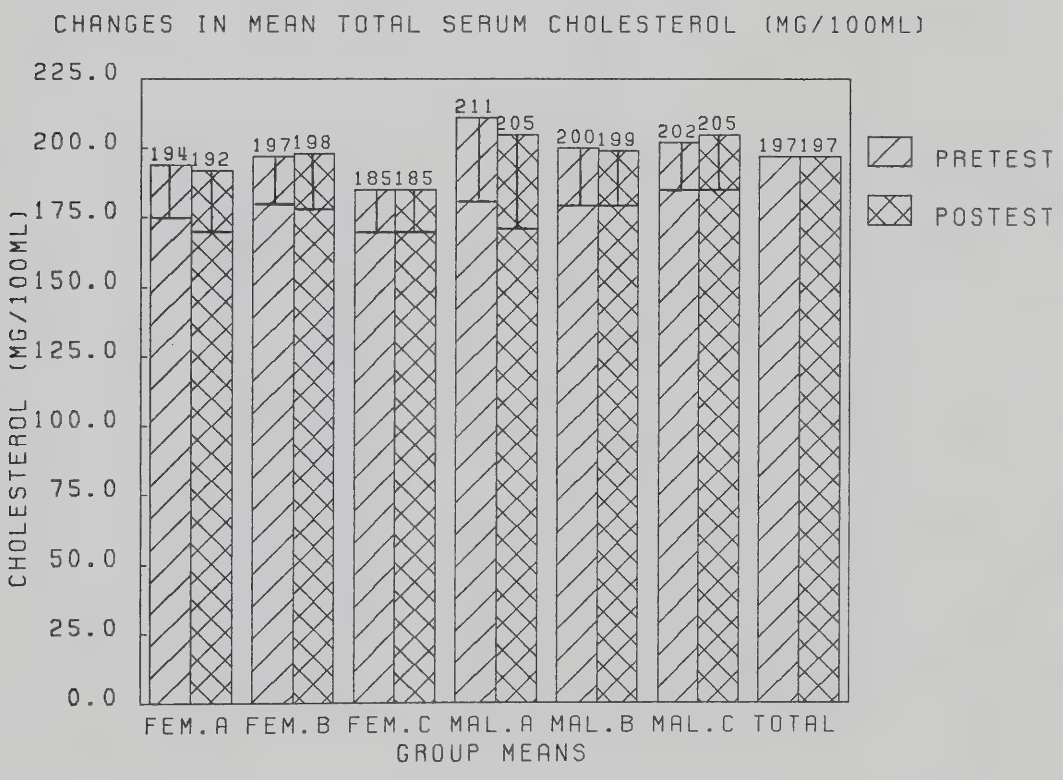
Total changes in mean serum HDL-Chol (Figure 9), LDL-Chol (Figure 10) and free cholesterol levels (Table 7) were insignificant. Even if there were some changes in mean groups pre and post test they were very small and not large enough to detect an important difference. There was not even a tendency that could make these results significant; for example, the HDL/LDL-Chol ratio (Figure 12). Data showed a higher ratio in the group FEM.C. This may be partly due to the sampling error and also to the lower mean age of that group. In group FEM.A and FEM.B we had more women in their menopause and this factor has been known to show a rapid ratio decrease (Kannel et al. 1976, LRC Data Book 1978). The actual data suggest similar findings to Allison et al. (1980). Identifying the APO AI and APO B may not add any more value in the present study since in the Kim and Kalkhoff (1979) study the only significant change in

HDL/LDL-Chol ratio was concomitantly related at the same time to a significant APO AI/APO B ratio.

Changes in mean VLDL-Chol (Figure 12) are shown to be insignificant as for the VLDL free cholesterol (Table 5). Even if group MAL.A and MAL.B showed a decrease in their mean values they are not close to the calculated critical mean difference of 2.07 at p.05. These insignificant changes were also observed in mean serum T.G. from 144.75 to 148mg% (Figure 13) and was this expected since the main part of circulating T.G. in a fasted subject was in VLDL-Chol. These results have been previously reported and were shown to be stable with an incressed of VO2 max (Gettman et al., 1976; Hunt and White, 1980; Allison et al., 1980). Although if any lipid change had to be significant T.G. should be the first, because it has the highest discriminant change with training (Hartung and Squires, 1978); Holloszy et al., (1964) reported a T.G. change from 208 to 125mg% with no concomitant decrease in serum cholesterol from a six month program of endurance training.

LCAT IRE (Figure 14) as well as LCAT activity (micro mol/li.hr) (Table 7) were not shown to be significant, but all the groups had an increase with time except for a slight decrease in MAL.C. In group MAL.A the mean difference in pre and post of 1.02 was even higher than the critical mean difference. Although this significance was lessened by the fact that there were only four subjects in that group, the different increase may be due just to the random assignment.

FIGURE IV.7



Critical mean difference when n=6/gr

5.09mg% at p.05 7.06mg% at p.01

⊥ =Standard deviation

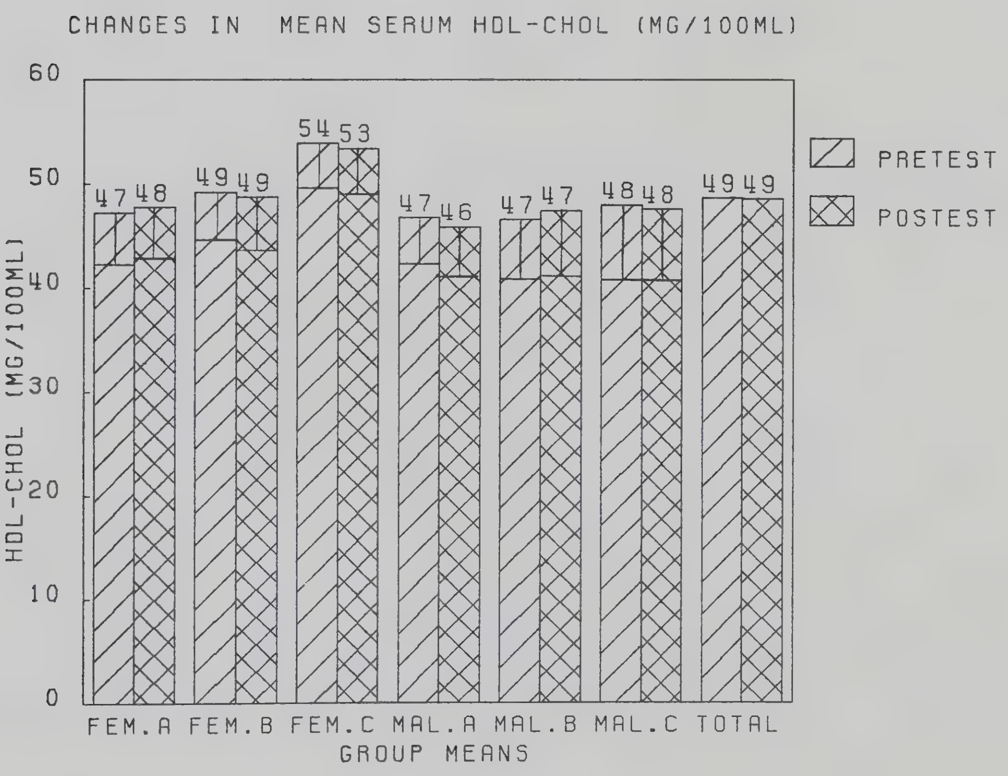
FIGURE IV.8

Critical mean difference when n=6/gr

2.71mg% at p.05 3.65mg% at p.01

⊥ =Standard deviation

FIGURE IV.9



Critical mean difference when n=6/gr

1.65mg% at p.05 2.21mg% at p.01

=Standard deviation

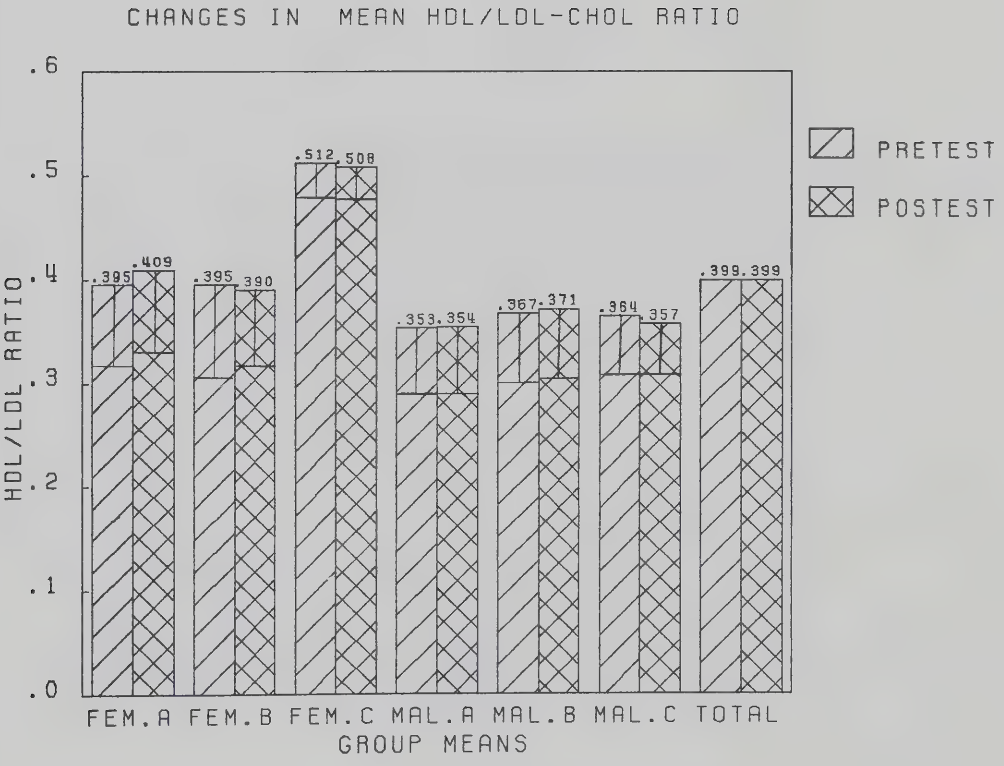
FIGURE IV.10

Critical mean difference when n=6/gr

3.87mg% at p.05 5.19mg% at p.01

=Standard deviation

FIGURE IV.11

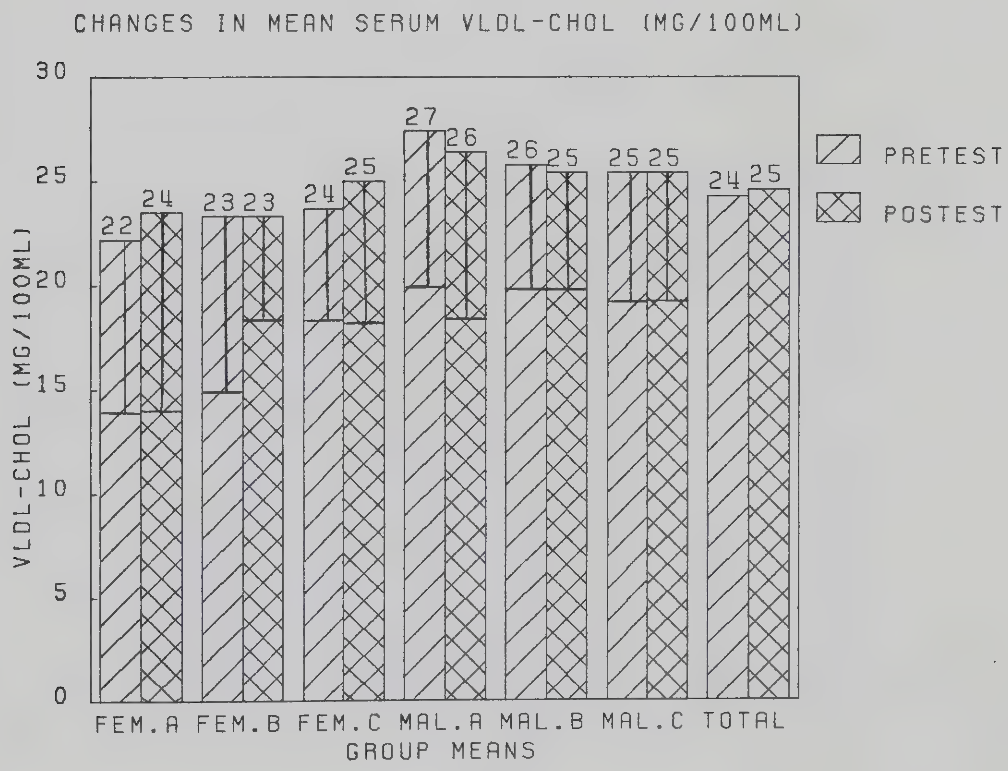


Critical mean difference when n=6/gr

.0257 at p.05 .0345 at p.01

⊥ =Standard deviation

FIGURE IV.12

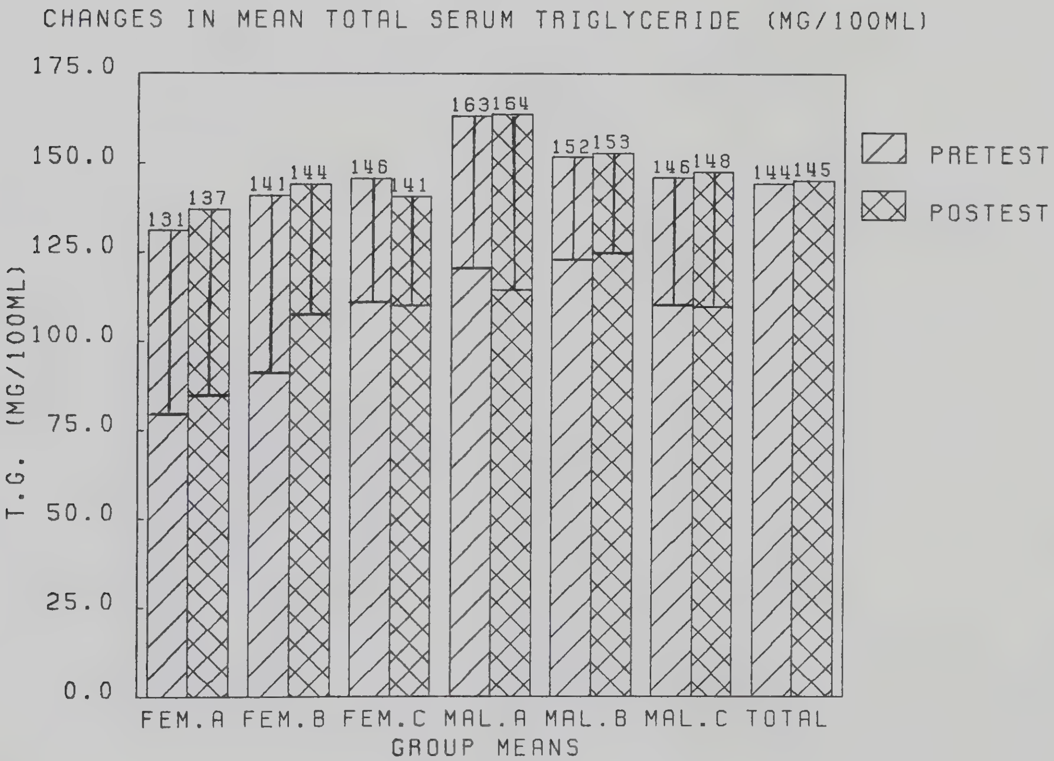


Critical mean difference when n=6/gr

2.07mg% at p.05 2.78 at p.01

=Standard deviation

FIGURE IV.13

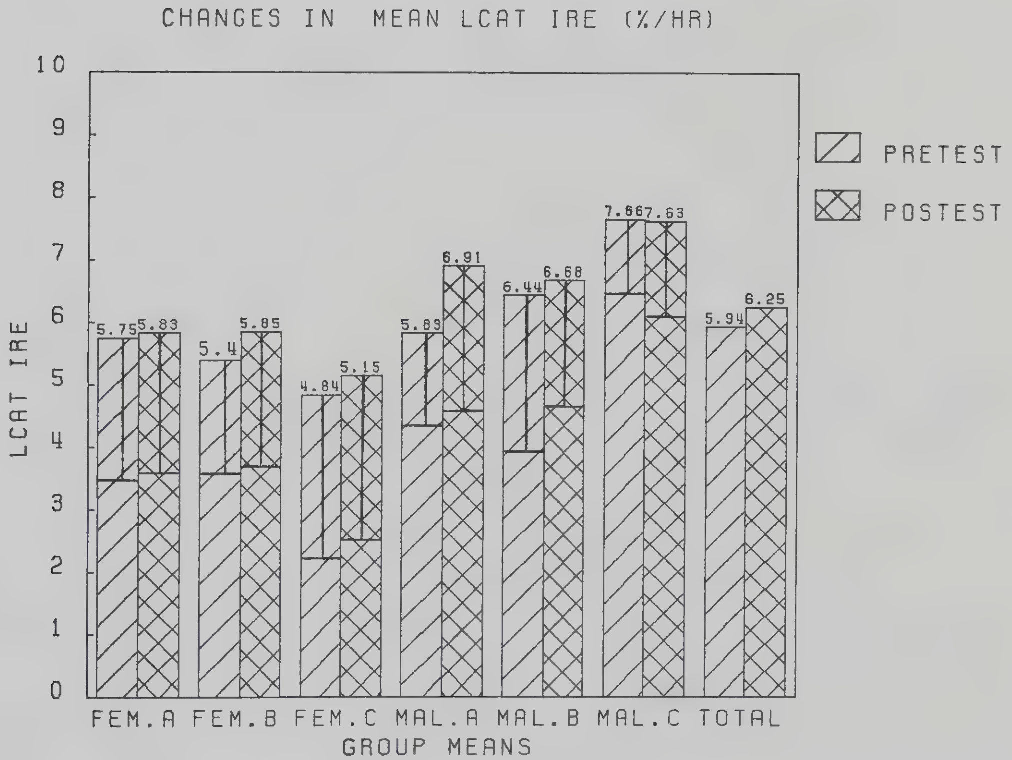


Critical mean difference when n=6/gr

22.48mg% at p.05 30.18mg% at p.01

⊥ =Standard deviation

FIGURE IV.14



Critical mean difference when n=6/gr

0.70 IRE(%/HR) at p.05   0.94 IRE(%/HR)at p.01

=Standard deviation

LCAT IRE= Lecithin cholesterol acyl transferase

initial rate of esterification

The changes in LCAT IRE with time can hardly be related to the changes in fitness since group FEM.C also had a mean increase, therefore the present tendency may be more due to experimental error or daily fluctuation (Schlierf et al., 1979).

H. General discussion

The response of several physiologic measures and blood parameters have been examined in this investigation. Some physiologic measures, such as post exercise heart rate and resting blood pressure were shown to change without a concomitant change in the serum lipids. Therefore the training subjects would have decreased their cardiovascular risk index as measured by a questionnaire, indicating the increase in their level of activity which was considered as an important factor. However, the present study showed that the changes in this lifestyle with an increase in fitness were not related to the metabolic parameter of cardiovascular disease. Although the subjects were normolipidics in mean cholesterol, their mean HDL/LDL-Chol ratios were fairly low compared to younger subjects (LRC Data Book, 1978; Kim and Kalkhoff, 1979) or trained subjects of their age group.

Therefore, the experimental population could change their cardiac risk index not only for a statistical significance but also as a physiological mean. For example, T.G. levels of 78mg% were observed in highly trained adults

(Hartung and Squires, 1978) while this study's subjects were at 144mg%. The actual mean of 48.5mg% in HDL-Chol was low compared to the 66mg% of the trained subjects of Hartung and Squires study (1978). Although total cholesterol seemed to be less susceptible to modification when highly trained subjects with a mean of 189mg% (from the same study) were compared with our males subjects of 205mg%, this high HDL-Chol level and lower T.G. with little change in total serum cholesterol have also been observed by Lehtonen and Viikari (1978a, 1978b). However, some others observed a concomitant decrease in total cholesterol with higher HDL-Chol (Wood et al., 1976). Finally the LDL-Chol of our males could been modified when their mean value of 132mg% was compared with 125mg% in trained subjects (Wood et al., 1976).

There were subjects who increased their fitness levels and it has been reported that these changes are related to metabolic parameters which are related in turn to the energy metabolism for muscular contraction (Holloszy, 1976). Although the serum lipids have very little to do with human physical work capacity, and it has been shown that HDL/LDL-Chol ratios are much higher in highly fit individuals. However it cannot be stated that a high HDL/LDL-Chol ratio will be metabolicaly related to high physical work capacity since that ratio can also be influenced by other means such as genetics, nutrition, age, sex, etc.... So, changes in lipoprotein level are more a

consequence of training than a need for better working capacity. Therefore attempts to modify lipoprotein levels by short term fitness improvement may have little to do with the individual's increase in working capacity as observed in the present study, but may be significantly related to the starting lipoprotein levels and secondly, to the intensity, frequency and duration of training. Unfortunately, changes in dietary habits and it's effect with training, although known to influence the serum lipids, cannot be substantiated by the present study since there were no significant changes in either dietary habits, serum lipids or body weight, as have been observed in other studies (Squires et al., 1980).

Many different groupings were possible in the present study since the present design did not show any substantial evidence on the effect of dietary information. Therefore one of the groupings that could lead to some understanding of the susceptibility of the changes in serum lipids was to see if those who increased HDL-Chol may have some related parameter to explain such changes.

A number of new groups were therefore formed. FEM.D (n=10) and MAL.D (n=5) were those with training and no increase in HDL-Chol. FEM.E (n=8) and MAL.E (n=6) were those with training who increased their HDL-Chol. FEM.C and MAL.C were the same controls as in the present design. The analysis of variance of the new grouping are shown in Appendix E while the new means are shown in table 8.

TABLE IV.8

Serum Lipids Grouping by HDL-Chol

GROUP	FEM.D	FEM.E	FEM.C	MAL.D	MAL.E	MAL.C	TOTAL
HDL-Chol mg%							
Pre.	48.28	48.40	53.88	46.26	46.82	47.93	48.61
S.D.	5.55	3.98	4.49	5.05	5.00	6.99	
Post	47.19	49.56	53.40	45.68	47.72	47.50	48.50
S.D.	5.58	4.25	4.47	5.79	4.85	6.58	
HDL/LDL-Chol ratio							
Pre.	0.383	0.410	0.512	0.365	0.358	0.364	0.399
S.D.	0.073	0.083	0.038	0.059	0.070	0.052	
Post	0.376	0.427	0.508	0.352	0.375	0.357	0.399
S.D.	0.072	0.093	0.029	0.054	0.068	0.051	
LDL-Chol mg%							
Pre.	128.6	120.8	105.7	129.2	133.2	132.1	125.0
S.D.	18.2	16.7	12.4	24.4	20.2	13.3	
Post	128.4	119.6	105.3	131.7	129.2	133.7	124.6
S.D.	19.3	20.5	10.9	21.3	17.5	13.2	
HDL/Total Serum Cholesterol ratio							
Pre.	0.243	0.256	0.292	0.233	0.228	0.237	0.249
S.D.	0.030	0.042	0.017	0.027	0.033	0.028	
Post	0.239	0.263	0.289	0.231	0.238	0.232	0.249
S.D.	0.032	0.044	0.019	0.033	0.030	0.027	
Total Serum Cholesterol mg%							
Pre.	199.3	190.9	184.9	201.0	206.3	202.4	197.2
S.D.	16.84	18.29	14.50	30.78	19.29	16.95	
Post	198.1	191.4	184.8	200.7	201.4	204.8	196.7
S.D.	17.84	21.77	14.45	33.26	15.32	16.53	
Total Physical Activity min/2wk							
Pre.	184.8	185.0	100.8	92.0	135.0	214.5	158.3
S.D.	167.8	168.7	128.9	60.4	126.2	144.5	
Post	329.6	216.4	70.7	431.8	285.3	90.8	240.6
S.D.	230.2	117.6	88.3	269.0	270.9	124.9	
Predicted VO2 Max ml/kg							
Pre.	26.61	27.25	31.03	35.20	34.55	35.00	30.82
S.D.	5.587	3.485	3.899	4.220	4.326	8.179	
Post	29.04	30.65	30.70	37.68	37.00	35.78	32.80
S.D.	4.877	5.019	3.738	4.338	4.486	8.164	
Total Daily Caloric Intake							
Pre.	1633	1888	2089	2102	2389	2318	2037
S.D.	431	337	405	623	308	559	
Post	1631	1977	2215	1944	2538	2400	2099
S.D.	354	499	403	521	548	761	
Percent Body Fat							
Pre.	30.49	32.14	31.33	27.18	22.78	28.18	29.06
S.D.	6.94	4.34	6.54	5.93	5.83	9.08	
Post	30.45	31.83	31.47	26.98	22.62	28.13	28.96
S.D.	6.76	4.23	6.79	5.83	5.85	8.78	

HDL-Chol showed a significant increase with group E in treatment by time (p.033) and obviously it comes from the bias grouping. But this new grouping becomes more powerful when the HDL/LDL-Chol ratio was calculated with a significant mean increase in FEM.E and MAL.E at p=.0000 level, which can be partially explained by the mean decrease in LDL-Chol of these groups (p=.078). A similar powerful change in mean increase of HDL/Total serum cholesterol for FEM.E and MAL.E (p=.0006) was seen, which were not followed by changes in total cholesterol. Those significant changes in lipids did vary distinctly from:

1.the total activity, where group FEM.E and MAL.E did not have a higher increase in activity rate;

2.the predicted VO2 max mean changes, where there was no distinctive increase from either training group;

3.the caloric intake showed no significant differences with time between training groups;

4.the percent body fat did not appear to be a factor related to such lipids changes.

These transformed groupings supported the notion that short term increases in fitness are not followed by lipids changes, and that total lipid values such as cholesterol, HDL-Chol and LDL-Chol are not the best discriminant factor to assess changes, while ratios of HDL/LDL-Chol and HDL/Total serum cholesterol are more appropriate and this can be observed also in Ready's data (1980). These new mean changes in HDL/LDL-Chol ratio in treatment by time were

barely close to the significant mean difference at p.05 in normal groupings (Figure 12) therefore providing evidence that changes in lipid levels in normal and a randomly distributed population were unlikely to show evidence of alteration from a short fitness program. The actual change in HDL-Chol in some subjects may be due to diurnal variations (Schlierf et al., 1979).

V. SUMMARY AND CONCLUSION

The effects of a popular fitness program was studied upon 41 sedentary adults; 8 female and 4 male were in a training group (A), 10 female and 7 male were in a training plus diet information group (B), and 6 female and 6 male acted as a controls group (C). Subjects were tested prior to the beginning of the program and at the end of the classes, i.e., 12 weeks after. The following dependent variables were analyzed: body weight and fat, systolic and diastolic blood pressure, three day dietary recall, post exercise heart rate, serum lipids, serum glucose and serum uric acid.

Significant decreases in post exercise heart rate, resting systolic and diastolic blood pressure were observed in the second test in group A and B. No significant dietary alteration and no changes in predicted percent body fat were noticeable in groups A, B, and C during the study. Serum analysis showed no significant changes in cholesterol, lipoproteins, LCAT, glucose and uric acid.

Within the limitations of the present experiment the following conclusions appear justified:

1. Twelve weeks of fitness training were sufficient to cause a significant decrease in post exercise heart rate.
2. Changes in total body weight, as in percent body fat

were not significantly modified.

3.Systolic and diastolic blood pressure were shown to decrease from the program.

4.Diet characteristics were not modified from the dietary information distributed during the study.

5.Sedentary adults may show willingness to get into fitness programs, but other changes in their lifestyle, such as diet, are more difficult to alter.

6.Serum lipids were not significantly modified by such programs.

7.Short term increases in fitness were not concomitantly related to serum lipid changes and vice versa.

8.Two known cardiac risk factors were modified from this fitness program, i.e., lower blood pressure and increased physical activity.

9.Men and women showed similar changes as a result of the fitness the program.

Several recommendations for futher investigation regarding fitness programs and reducing the cardiac risk index can be made:

1.Pamphlet distribution at large, as in this study, should be modified by a good explanation before distribution and the educational component of fitness classes should be more than informative for short term changes.

2. Different pamphlets may be more influential by their content or their approach, i.e., for example making pamphlets that are challenging the weaknesses of the individual may be more positive.

3. To relate changes in serum lipids with improvement in fitness the duration of the program should be longer than twelve weeks.

4. Physical activity as a cardiac risk index should be accounted for by a time factor.

5. Changes in fasting lipoprotein levels from highly fit and low fit individuals should be investigated to ascertain if it is the result of acute exercise, or if in fact a difference in the rate of post prandial lipid absorption, or lipid clearance or synthesis may be involved.

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VII. APPENDIX A

A. Subject informed consent

I desire to engage voluntarily in a physical fitness program to see the effects upon my working capacity, body fat, blood pressure and selected blood parameters. The training program will last between 12 to 15 weeks; two times a week a YMCA instructor will organise the session while the third session will be undertaken without supervision. In each session there will be a period of twenty to thirty minutes of aerobic work.

The testing procedures will be done after a 12 hour fast, a 72 hour alcohol restriction and I am asked to refrain from exercise for 12 hours before the test. First, systolic and diastolic blood pressure will be taken: second ten milliliters of blood will be drawn (from the antecubital vein); third, body weight will be recorded in kilograms; fourth, body fat will be measure by a skinfold caliper on the triceps, biceps, abdominal and suprailiac; fifth, a physical work capacity test.

I understand that I will step on double 20.3 cm steps at speeds identified for my age group. During the performance of the test my heart rate will be measured prior to and at the completion of the test.

I will first be given a three minute warm-up exercise at a rate equilivalent to 65 to 70% of the average aerobic power anticipated in a person in a 10 year older age group than mine. If a predetermined heart rate is not exceeded, I will then exercise for a further three minutes at 65 to 70%

of the average aerobic power for a sedentary person of my own age. If again my predetermined heart rate is not exceeded, I will exercise for a further three minutes at 65 to 75% of the average aerobic power for an individual ten years younger than I am. The test will be discontinued when I reach a predetermined heart rate or if I become distressed in any way or develop any abnormal response, wichever of the above occurs first. Every effort will be made to conduct the test in such a way as to minimize discomfort and risk. However, I understand that just as with other types of fitness tests there are potential risks. These include episodes of transient lightheadedness, fainting, chest discomfort, leg cramps and nausea.

Finally, a questionnaire to identify my caloric intake for the last three days shall be completed.

Any time before, during or after this period of testing I shall be able to ask questions concerning procedures or other aspects of the project. At any time I will be able to withdraw consent and to discontinue participation in the project or some of its parts without prejudice. In the questionnaire I am free to refuse to answer specific items of questions. I will be encouraged to follow the recommendations (verbal or written) from this project but any recommendation will not compulsory.

All the data and information of this study will be confidential, however, the results may be used for statistical or scientific purposes with my right of privacy

retained.

N.B. For those who served as control subjects, they are asked to continue their normal seasonal activity without engaging in a fitness program.

I have read the foregoing and I understand it. Any question which have arisen or occured to me have been answered to my satisfaction.

SIGNED......................DATE...................

NAME........................

B. PAR Q & YOU

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate of those who should have medical advice concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read them carefully and write YES or NO opposite the question if it applies to you.

1. Has your doctor ever said you have heart trouble?

2. Do you frequently have pains in your heart and chest?

3. Do you often fell faint or have spells of severe dizziness?

4. Has a doctor ever said your blood pressure was too high?

5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?

6. Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?

7. Are you over age 65 and not accustomed to vivorous exercise?

<u>YES to one or more questions</u>.

If you have not recently done so, consult with your personal physician by telephone or in person before increasing your physical activity and/or taking a fitness test. Tell him what questions you answered yes on PAR-Q, or show him your copy. After a medical evaluation, seek advice from your physician as to your suitability for: unrestricted physical activity, probably on a gradually increasing basis. Restricted or supervised activity to meet your specific needs, at least on an initial basis. Check in your comunity for special programs or services.

<u>NO to all question</u>.

If you answered PAR-Q accurately, you have reasonable assurance of your present suitability for : A graduated

exercise program A gradual increase in proper exercise promotes good fitness development while minimizing or eliminating discomfort. An exercise test - A simple tests of fitness (such as the Canadian Home Fitness test) or more complex types may be undertaken if you so desire.

Postpone.

If you have a temporary minor illness, such as a common cold.

C. Your activities

Name:...................

a. During the last two weeks, how many times did you do any of the following exercises, sports or recreational activities?

b. About how much time did you spend on each occasion?

Walking (inclucing to and from work or school)

Jogging or running

Calisthenics

Bicycling (including to and from school)

Vigorous dancing

Skating

Skiing (downhill, crosscountry)

Racquet sports (tennis, badminton, squash, racquetball)

Other team sports (hockey, basketball, football, soccer, volleyball)

Swimming

Other (please specify)

D. Dietary recall

Name........................

1. Record your dietary intake for three consecutive das (include Sunday, Monday and Tuesday).

2. Description of food items should be as complete as possible:

a) Type of food (raw, cooked, frozen, etc.)

b) Method of preparation (baked, broiled, fried, etc.)

c) Brand name (if this affects nutrients composition)

d) Ingredients in recipes

e) Best estimation of compositon of commercial foods if this is not known

f) Amount of food (household measure and weight in grams)

g) Type of milk (whole, 2% of skim, etc...)

h) The cut of meat, lean or regular hamburger, etc...

<u>EXAMPLE</u>

8:00 a.m. poached egg, 1 large, 50grams.

8:00 a.m. 100% whole wheat bread, 2 slices, 50 grams.

8:00 a.m. margerine, 2 tsps., 10 grams.

8:00 a.m. orange juice (made from frozen concentrate), 8oz., 247 grams.

10:00 a.m. coffee - instant (regular dry powder), 1tbsp., 2

grams.

10:00 a.m. commercial chocolate chip cookies, 2" diameter, 17 grams.

10:00 a.m. milk - 2%, 4oz., 122 grams.

10:00 a.m. sugar (white granulated), 1tsp., 4 grams.

VIII. APPENDIX B

A. Pamphlet distribution

Before each reference the date of distribution is indicated with the group. If the subject was absent that night the pamphlet was mailed to him.

September 17, diet information and training group, Alberta Fitness "A Few Words About Fitness,"<u>Alberta Fitness</u>, Alberta Recreation and Parks, Edmonton, Alberta.

September 17, diet information and training group, Alberta fitness, "Jogging the Right Way," <u>Alberta Fitness</u>, Alberta Recreation and Parks, Edmonton, Alberta.

September 17, diet information and training group, Alberta Fitness, "Keeping Fitness in the Family,"<u>Alberta Fitness</u>, Alberta Recreation and Parks, Edmonton, Alberta.

September 17, diet information, Alberta Social Services and community Health, Why Weight," <u>Government of Alberta</u>, Edmonton, Alberta.

September 17, diet information, American Heart Association, "Your Diet and Your Heart," From Scriptographic Booklet, by Channing L. Bete Co., Inc., Greenfield, Mass., 1979.

September 17, diet information, Health and Welfare Canada, "Nutrient Value of Some Common Foods," Minister of Health and Welfare pub., Ottawa, Ont., 1979.

September 24, diet information and training, Alberta Fitness, "Physical Fitness...? Who Needs It!...A Case for Regular Exercise," <u>Alberta Fitness</u>, Alberta Recreation and Parks, Edmonton, Alberta.

September 24, diet information and training, Alberta Fitness, "Surprising...What You Don't Know About Fitness," <u>Alberta Fitness</u>, Alberta Recreation and Parks, Edmonton, Alberta.

September 24, diet information and training, American Heart Association, "E is for Exercise," Am. Heart. Assoc., Dallas, Texas, 1977.

September 24, diet information, American Heart Association, "Save Food $$ and Help Your Heart," Am. Heart Assoc., Dallas, Texas, 1979.

September 24, diet information, American Heart Association, "The Way to Man's Heart," Am. Heart Assoc., Dallas, Texas, 1972.

September 24, diet information, American Heart Association, "You and Your Blood Pressure," from Scriptographic Booklet, by Canning L. Bete Co., Inc., Greenfield, Mass., 1979.

September 24, diet information and training, Smith, R., How to stay fat, and enjoy it! Reader's Dig., 215-218, Nov., 1980.

September 24, diet information and training, Zuti, W.B., Fat and fitness questions with thin answers, Fitness Fantasia Fanfare, 1(5): 1-2, 1981.

October 15, diet information, Alberta Social Services and Community Health, "Nutrition When You're on Your Own," Government of Alberta, Edmonton, Alberta.

October 15, diet information, American Heart Association, "Recipes for Fat Controlled Low Cholesterol Meats." Am. Heart Assoc., Dallas, Texas.

Ocotber 15, diet information American Heart Association, "Weight Reduction," Am. Heart Assoc., Dallas, Texas.

October 15, diet information, American Heart Association, "You and Your Heart," by Channing L. Bete, Inc., Greenfield, Mass., 1979.

October 15, diet information and training, Fitness and Amateur Sport Canada, "Aerobic Fitness," from the Government of Canada, Ottawa, 1979.

October 15, diet information and training, Fitness and Amateur Sport Canada, "Lunch on the Run," from the Government of Canada Fitness and Amateur Sport, Ottawa, 1978.

October 15, diet information and training, Fitness and Amateur Sport Canada, "Physical Activity and Weight Control," from the Government of Canada Fitness and Amateur Sport, Ottawa, 1978.

October 15, diet information and training, Fitness and Amateur Sport Canada, "You and Your Heart Rate," from the Government of Canada Fitness and Amateur Sport, Ottawa, 1978.

October 15, diet information, Health and Welfare Canada, "Canada's Food Guide Handbook," from the Minister of Supply and Services Canada, Ottawa, 1979.

October 15, diet information, Health and Welfare Canada, "Food and Your Heart," from Health and Welfare Canada, Ottawa.

October 15, diet information, Health and Welfare Canada, "Shopping for Food and Nutrition," from Information Services, Agriculture Canada, Ottawa, 1980.

IX. APPENDIX C

PROCECURES FOR SERUM ANALYSIS

A. Cholesterol and esterified cholesterol

Principle

Esterified cholesterol plus potassium hydroxide gives cholesterol plus fatty acid. Cholesterol plus cholesterol oxidase plus oxygen gives delta-four-cholestenone plus hydrogen peroxide, exibiting maximum abserbance at 240nm.

Instrument

Spectrophotometer capable of measurement at 240nm.
Water bath at 37 degrees Celsius
Centrifuge

Reagents

Sodium dihydrogen phosphate, $NaH_2PO_4.H_2O$.
Disodium hydrogen phosphate, Na_2HPO_4.
Absolute ethanol.
Polyoxyethylene 9 lauryl ether, from Sigma Chem. Co.
Potassium hydroxide, KOH.
Cholesterol oxidase from Nodardia erythropolis, from Sigma Co.
One molar Amonium sulfate solution, $(NH_4)_2 SO_4$

Preparation of Solutions

Phosphate buffer (pH 7.5): dissolve 60.5g of $NaH_2PO_4.H_2O$, 10.2g of Na_2HPO_4, and 4g of polyoxyethylene 9 lauryl ether in water up to 1,000ml.

Cholesterol oxidase (6.25U/ml):dilute stock suspension as

required with 1M amonium sulfate solution.

Potassium hydroxide solution: dissolve 33g of KOH in water and make up to 100ml (final concentration 33%w/v).

Procedures

Hydrolysis of esterified cholesterol: Introduce 0.5ml sample, 4.5ml ethanol, and 0.3ml potassium hydroxide solution into stoppered test tubes and swirl around carefully. Place the stoppered test tubes in a water bath at 37 degrees Celcius for 60 min. Centrifuge the suspension 10 min 3000'g) and use the clear surpernatant for the determination.

Dilute 0.2ml hydrolysate (to determine the total cholesterol) of 0.05ml sample (to determine the free cholesterol) with 10ml phosphate buffer: use 3.5ml in the determination for sample and sample blank.

Pipette 0.02ml of cholesterol oxidase solution into the bottom of the test tube and add the diluted sample. This is sample A. Pipette 0.02ml of water into the bottom of the test tube and then add the diluted sample. This is sample blank B. Mix, allow to stand for about 15 min. at 20-25 degrees Celsius. For the measurements, pour the sample into cuvette A and the sample blank into cuvette B.

Set extinction of cuvette B to zero, and measure extinction of cuvette A. The extinction E sample is obtained.

Reagent blank: Pipette into the bottom of test tube 0.02ml of cholesterol oxidase solution and add 3.5ml of phosphate buffer. This is sample A. Pipette 0.02ml of water into the

bottom of the test tube and add 3.5ml of phosphate buffer. This is sample B. Mix, pour the sample into appropriate cuvette, set extinction of cuvette B to zero and measure extinction of cuvette A. The extinction E Blank is obtained. E sample minus E blank = delta E, is used in the calculation

Total cholesterol (mg/ml)= delta E x 13.6

Free cholesterol (mg/ml)= delta E x 5.04

Cholesterol (in absolute ethanol) levels equivalent to concentrations of 25, 59, 75, 100, 200 and 300mg/100ml yielded a correlation coefficient of 0.99.

A coefficient of variation of 3.34% for free cholesterol and 2.8% for total cholesterol was obtained for 10 replicate assays of serum sample.

Nota Bene: this method was used for the determination of cholesterol and esterified cholesterol in serum, in VLDL-Chol, LDL-Chol, HDL-Chol and LCAT activity.

B. Glucose in serum

Principle:

Glucose plus O-Toluidine gives a colored complex, exibiting maximum absorbance at 635nm.

Instrument

Spectrophotometer capable of measurement at 635nm.

Water bath at 100 degrees Celsius.

Reagents

O-Toluidine reagent, stock No 635-6 from Sigma Chem Co. (contains O-Toluidnine 6% w/v in glacial acetic acid.

Glucose standard solution, stock No 635-100 from Sigma Chem. Co. Standardized at 100mg/100ml with benzoic acid added as preservative.

Procedure

Label three or more test tube: blank, standard, test 1, test 2, etc.

To blank add 0.1ml water. To standard add 0.1ml glucose standard solution (stock No 635-100). To test add 0.1ml serum.

To each test tube add 5.0ml O-Toluidine reagent (stock No 635-6), mix by lateral shaking.

Put all tubes into a vigorously boiling water bath for exactly ten minutes.

Quickly remove all tubes and cool to room temperature by placing in tap water for approximately three minutes.

Transfer contents of tubes to cuvets and read absorbance of standard and test at 635nm, using blank as reference.

Complete readings within thirty minutes.

Calculation: glucose(mg/100ml)=test/standard x 100.

A coefficient of variation of 5.4% was obtained for 10 replicate assays of a serum sample. A glucose levels equivalent to concentrations of 0, 50, 100, 150, 200 and 250mg/100ml yielded a correlation coefficient of 0.99.

C. Lecithin cholesterol acyl transferase

Principle

The enzymatic decrease of free cholesterol content in serum is caused by the LCAT reaction.

Instrument

Spectrophotometer capable of measurements at 240nm.

Water bath at 37 degrees Celsius.

Reagents

Identical to those used in the determination of free cholesterol.

Procedure

Pipette 0.05ml of sample into the bottom of the test tube and incubate 40min. at 37 degrees Celcius. Measure absorbance at 240nm as described in the method of cholesterol determination. This is absorbance E2.

Pipette 0.05ml of sample into the bottom of the test tube and measure absorbance at 240nm as described in the method of cholesterol termination. This is absorbance E1.

Calculation: initial rate of esterification (IRE)

IRE = (E1 - E2)/E1 X 150(%/hr)

LCAT nmol/li/hr = IRE(%/hr) X Total Free Chol.(mg%) X 130/5.04 Where 130/5.04 is a constant for transforming the level of cholesterol in mg% to nmol.

A coefficient of variation of 8.1% was obtained for 10 replicate assays of a serum sample.

D. Lipoprotein: LDL-Chol and VLDL-Chol

Principle

Each class of lipoprotein has a different density, thus each lipoprotein will float on his gradient density.

Instrument

Spectrophotometer capable of measurements at 240nm.

Water bath at 37 degrees Celsius.

Centrifuge.

Ultracentrifuge: model used was a Beckman (Model L) with a type 50 fixed head rotor.

Reagent

Sodium choloride NaCl.

Ethylenediamine tetraacetic acid, EDTA.Na2.

Sodium hydroxide, 1N. NaOH.

Sodium bromide, NaBr.

Preparation of solutions

Density 1.006 gm/ml solution: 11.40 gm of NaCl and 0.1 gm of EDTA.Na2 are added, and the solids are dissolved by mixing, The flask is filled to volume and three ml of additional water are added.

Density 1.182 gm/ml: 24.98 gm of NaBr are added to 100 ml of the above density 1.006 solution.

Procedure

Pipette two ml of serum into ultracentrifuge tubes. One milliliter of density 1.006 solution is layered over the surface. The tubes are centrifuged in the rotor at 20,000rpm (26,000g) without refrigeration for 30 minutes. This is spin

one, and a fraction which may operationally be defined as "chylomicrons and particules" is floated to the top of the tube.

A Pasteur pipette is used to removed the top one ml. from the first centrifuge tubes.

The two ml. infranatant solutions from spin one are again overlaid with one ml of density 1.006 solution and centrifuged 16 hours at 41,200 rpm (114,000g) with refrigeration of 4 degrees Celsius. This is spin two and the top one ml contains lipoproteins VLDL-Chol which is removed by a Pasteur pipette.

The bottom two ml of spin two are mixed to disperse the gelatinous material at the bottom of the tubes, because this is rich in lipoproteins. One ml of density 1.182 solution is added and the tube is mixed. The tubes are centrifuged 20hours at 41,000rpm (114,000g) with refrigeration of 4 degrees Celsius. This is spin three, and its top one ml contains the LDL-Chol fraction.

The level of cholesterol and esterified cholesterol are measured as described in part A of this appendix.

E. Lipoprotein HDL-Chol

<u>Principle</u>

Heparin and manganese chloride ($MnCl_2$) are added directly to the serum to precipitate LDL-Chol and VLDL-Chol leaving HDL-Chol in solution.

Instrument

Spectrophotometer capable of measurements at 240nm.

Water bath at 37 degrees Celsius.

Centrifuge.

Reagents

Heparin from porcine intestinal mucosa from Sigma Chem. Co.

Sodium chloride, NaCl.

Manganous chloride, MnCl2.

Preparation of solutions

Sodium chloride solution (0.15M): dissolve 0.877gr. of NaCl in water and make up to 100ml.

Heparin: dilute heparin with 0.15M NaCl, final concentration 5,000U/l.

Manganous chloride: dilute manganous chloride with water, final concentration 92mmol/l.

Procedure

Pipette 0.5ml of serum into a small test tube. Add 0.025ml of heparin (5,000U/ml) and mix well. Then add 0.025ml of MnCl2 solution and mix immediately.

Let the prepared samples stand at room temperature for 30 minutes. Then centrifuge at 800g for ten minutes at 4 degrees Celsius.

The clear supernatant is the HDL-Chol solution.

The level of cholesterol and esterified cholesterol are measured as described in part A of this appendix.

This method had shown level values of 3.3% higher than those of recovered sample sample after spin three of

LDL-Chol.

F. Triglyceride

Principle

Triglycerides are extracted from serum with isopropanol. Interfering substances, including glucose, phosphatides and bilirubin are removed by a solid absorbant. The triglyceride-containing extract is then subjected to the following reactions:

1.Triglycerides plus KOH gives glycerol plus fatty acid.

2.Glycerol plus periodate gives formaldehyde.

3.Formaldehyde plus NH4+ plus acetylacetone gives diacetyldihydrolutidine.

The final product, diacetyldihydrolutidine, is yellow, exhibiting maximum absorbance at 410nm.

Instrument

Spectropholometer capable of measurements at 410nm.

Water bath at 60 degree Celcius.

Centrifuge.

Reagents

Triglyceride purifier, stock No 405-8, preweighed vial containing 0.8gr of activated alumina, from Sigma Chem. Co.

Isopropanol anhydrous, stock No 405-7, from Sigma Chem. Co.

Triolein Standard, stock No 405-10, contains 300mg triolein dissolved in 100ml anhydrous isopropanol, from Sigma Chem. Co.

Potassium hydroxide, 1N, stodk No 405-1, from Sigma Chem.

Co.

Acetic acid solution, 2N, stock No 405-12, from Sigma Chem. Co.

Sodium m-Periodate in preweighed vial of 125mg, stock 405-9, from Sigma Chem. Co.

Ammonium acetate solution ,2M, stock No 405-2, from Sigma Chem. Co.

Acetylacetone, stock No 405-4, from Sigma Chem. Co.

Preparation of solutions

Periodate solution, prepare by reconstituting a vial of sodium m-periodate, stock No 405-9, with 50ml acetic acid solution, stock No 405-12.

Color reagent, prepare by mixing: 20ml ammonium acetate solution (stock No 405-2), 40ml isopropanol anhydrous (stock No 405-7), 0.15ml acetylacetone (stock No 405-4). Aging is required, by permitting the color reagent to stand overnight.

Procedure

I. Extraction

Label three of more triglyceride purifier preweighed vials (stock No 405-8) as follows: blank, standard, test 1, test 2, etc.

To blank add: 5.0ml isoporpanol (stock No 405-7) and 0.2ml water. To standard add: 4.8ml isopropanol (stock No 405-7), 0.2ml water and 0.2ml triolein standard (stock No 405-10). To test add: 5.0ml isopropanol (stock No 405-7) and 0.2ml serum. Swirl tube gently while adding.

Shake with a mechanical mixer, or manually, for least 5 minutes (longer time not harmful). Allow vials to stand for a few seconds until absorbant starts to settle. To facilitate separation of fluid from solids in the following step, give each vial a single sharp snap with the wrist while grasping the capped vial from the top. This will tend to wash most of the solids down to the bottom of the vial. Centrifuge at about 3,000 rpm for 5 minutes at 4 degrees Celsius to obtain a clear supernatant.

II. Saponification

Label three or more screw-cap tubes as follows: blank, standard, test 1, test 2, etc.

Carefully transfer 2.0ml of clear supernatant to the correspondingly labeled tube. Into each tube, pipette just above the liquid level: 0.5ml 1N KOH. Mix by swirling. Incubate all tubes 60 degrees Celcius for five minutes. Remove tubes from water bath and cool to room temperature with tap water for 2-3 minutes.

III. Oxidation

To each tube, add 0.5ml periodate solution, mix immediately after each addition. Start timer after each addition to the first tube and note interval between additions.

IV. Color development

Ten minutes after addition of periodate solution to the first tube add to each tube: 3.0ml color reagent this should be done at approximately the same rate at which the periodate solution was added. Mix each tube after every

addition.

Cover tubes and place in 60 degree Celsius water bath for 30 minutes (srew-cap should be loosened to prevent pressure increase during incubation).

Remove tubes from water bath and cool to room temperature with tap water for 2-3 minutes.

Transfer contents of tube to correspondingly labeled cuvets.

Set blank to zero and read absorbance (A) of standard and test at 410nm.

Calculations: Triglycerides (mg/100ml) equal A test/ A standard X 300.

A coefficient of variation of 5.4% was obtained for 10 replicate assays of a serum sample. A triglyceride levels equivalent to concentrations of 0, 75, 150, 225, 300 and 375mg/100ml yielded a correlation coefficient of 0.99.

G. Uric acid

<u>Principle</u>

Uric acid undergoes oxidation to allantoin through the action of uricase. The decrease in absorbance at 292nm is proportional to uric acid concentration.

<u>Instrument</u>

Spectrophotometer capable of measurements at 292nm.

<u>Reagents</u>

Glycine buffer solution, 0.6mol/li, pH 9.4, stock No 292-6 from Sigma Chem Co.

Uricase enzyme suspension (hog liver) in ammonium sulfate

solution, activity 0.2-0.4U/ml (stock No 292-8), from Sigma Chem. Co.

Procedure

Pipette into a test tube 0.2ml of serum, 1.0ml of glycine buffer solution (stock No 292-6), 6.0ml water and mix well. Pipette into each of two test tubes: 3ml of mixture prepared in the previous step. Label one blank and the other test. Pipette into blank 0.05ml of water and mix well. Pipette into test 0.05ml of uricase enzyme stock No 292-8, and mix well. Allow both to stand at room temperature for 15 minutes.

Set spectrophotometer to wavelength of 292. With the blank in the lightpath adjust the instrument to read 0.400 absorbance. Place the test in lightpath and record its absorbance. Wait approximately five mimutes. Again adjust instrument to read 0.400 with the blank in the lightpath. Record the absorbance of the test again if absorbance has decrease, repeat reading intervals until constant. Record this reading as final absorbance.

Calculations: Delta A equal 0.405 minus final absorbance, and serum uric acid (mg/100ml) is equal to delta A by 50.

A coefficient of variation of 6.3% was obtained for 10 replicate assays of a serum sample.

X. APPENDIX D

A. Subjects training

Each subject had to keep a weekly record of his activity in the YMCA attendance book. The change in activity rate was determined by the questionnaire "Your Activities" (appendix A).

A typical lesson of one hour included a period of warm up, flexibility, muscular strength and endurance, aerobic exercise, recreational games and a cool down as described in the YMCA Shape Up Fitness Instructor's Manual For Canadians. Many times during the lesson the subjects were asked to check their heart rate in order to be within their target zone.

The warm up included: walk (continuous, on toes, heels and sides of feet), neck circles, arm circles, trunk circles, fencer's lunge, and light jogging.

The flexiblity included: side bends, hamstring stretch, shoulder extension and calf stretch.

The muscular strength and endurance included: push ups, sitting knee trucks, jumping jacks, chest raises, side raises. The first three parts last 15 to 25 minutes, and also included lifestyle information and discussion.

The aerobic component included jogging and recreational games for 20 to 35 minutes.

Finaly the cool down component included: walk, neck circles, shoulder circles, standing knee lift, alternate toe toucn, lie on floor and relax. The cool down period was 10 to 15 minutes.

The heart rate during a lesson period was checked by an "exercimeter" and it was observed that the subjects had an heart rate within their target zone during the aerobic component.

In the first two lessons a special emphasis was put on the knowledge of aerobic fitness, the measurement of pulse rate, the target heart and having them obtain their own values. The aerobic component of these classes lasts about 15 minutes and was mainly composed of walk-jog activities. In the following two weeks, the aerobic component increased to about ten minutes jogging and ten minutes of aerobic games. From the fourth week, the aerobic component included a minimum of fifteen minutes of jogging and ten minutes of games. From the ninth week, the aerobic exercise lasted about twenty minutes and the aerobic games fifteen minutes. During these activities the subjects were often asked to check their heart beat and it was observed that about 15 percent of the class was below or higher than their target heart rate, but it was not always the same subjects who were off target.

XI. APPENDIX E

S.=SEX T.=TIME Tr.=TREATMENT

A. Analysis of variance normal grouping

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
Total body weight					
SEX	5159.8	1	5159.8	14.64	0.0005
TREATMENT	788.8	2	394.4	1.12	0.3378
S.Tr.	584.9	2	292.4	0.83	0.4444
ERROR	12328.6	35	352.2		
TIME	0.107	1	0.107	0.07	0.7998
T.S.	0.323	1	0.323	0.20	0.6597
T.Tr.	0.121	2	0.060	0.04	0.9638
T.Tr.S.	1.857	2	0.928	0.57	0.5732
ERROR	57.490	35	1.642		
Percent body fat					
SEX	522.3	1	522.3	6.01	0.0193
TREATMENT	93.9	2	46.9	0.54	0.5872
S.Tr.	40.7	2	20.3	0.23	0.7924
ERROR	3039.2	35	86.8		
TIME	0.234	1	0.234	0.86	0.3608
T.S.	0.055	1	0.055	0.20	0.6542
T.Tr.	0.379	2	0.189	0.69	0.5063
T.Tr.S.	0.056	2	0.028	0.10	0.9028
ERROR	9.554	35	0.273		
Total activity min/2wk					
SEX	6156.2	1	6156.2	0.13	0.7172
Tr.	211788.2	2	105894.1	2.29	0.1158
S.Tr.	48801.3	2	24400.6	0.53	0.5941
ERROR	1615621.5	35	46160.6		
TIME	131836.4	1	131836.4	8.58	0.0060
T.S.	17881.0	1	17881.0	1.16	0.2882
T.Tr.	244446.7	2	122223.3	7.95	0.0014
T.Tr.S.	57607.0	2	28803.5	1.87	0.1686
ERROR	537987.0	35	15371.1		

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
Post exercise heart rate					
SEX	547.4	1	547.4	1.48	0.2325
TREATMENT	47.2	2	23.6	0.06	0.9384
S.Tr.	1401.9	2	700.9	1.89	0.1661
ERROR	12979.2	35	370.8		
TIME	3890.3	1	3890.3	85.28	0.0000
T.S.	50.8	1	50.8	1.11	0.2986
T.Tr.	1411.5	2	705.8	15.47	0.0000
T.Tr.S.	194.2	2	97.1	2.13	0.1342
ERROR	1596.6	35	45.6		
Predicted VO2 MAX					
SEX	776.3	1	776.3	15.63	0.0004
TREATMENT	130.1	2	65.1	1.31	0.2827
S.Tr.	47.2	2	23.6	0.48	0.6257
ERROR	1738.7	35	49.7		
TIME	65.15	1	65.15	59.31	0.0000
T.S.	0.01	1	0.01	0.01	0.9426
T.Tr.	24.54	2	12.27	11.17	0.0002
T.Tr.S.	3.97	2	1.98	1.81	0.1793
ERROR	38.45	35	1.10		
Systolic blood pressure					
SEX	1029.2	1	1029.2	5.73	0.0222
TREATMENT	1126.3	2	563.1	3.13	0.0560
S.Tr.	174.7	2	87.3	0.49	0.6192
ERROR	6290.9	35	179.7		
TIME	117.8	1	117.2	5.68	0.0227
T.S.	5.7	1	5.7	0.27	0.6038
T.Tr.	138.7	2	69.4	3.34	0.0469
T.Tr.S.	84.1	2	42.0	2.03	0.1469
ERROR	725.9	35	20.7		
Diastolic blood pressure					
SEX	780.8	1	780.8	9.48	0.0040
TREATMENT	167.1	2	83.5	1.01	0.3733
S.Tr.	249.6	2	124.8	1.51	0.2339
ERROR	2884.1	35	82.4		
TIME	294.2	1	294.2	28.77	0.0000
T.S.	5.9	1	5.9	0.57	0.4537
T.Tr.	59.1	2	29.5	2.89	0.0692
T.Tr.S.	36.4	2	18.2	1.78	0.1834
ERROR	357.9	35	10.2		

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
Total calorie intake					
SEX	2674712	1	2674712	7.80	0.0084
TREATMENT	2462445	2	1231222	3.59	0.0381
S.Tr.	364019	2	182009	0.53	0.5926
ERROR	11994896	35	342711		
TIME	43615	1	43615	0.39	0.5358
T.S.	10275	1	10275	0.09	0.7633
T.Tr.	143926	2	71963	0.65	0.5063
T.Tr.S.	38352	2	19176	0.17	0.8427
ERROR	3903220	35	111520		
Total fat intake					
SEX	2853.3	1	2853.3	2.53	0.1208
TREATMENT	5375.1	2	2687.5	2.38	0.1072
S.Tr.	2147.4	2	1073.7	0.95	0.3960
ERROR	39497.2	35	1128.5		
TIME	225.5	1	225.5	0.40	0.5287
T.S.	11.8	1	11.8	0.02	0.8851
T.Tr.	1989.4	2	994.7	1.79	0.1825
T.Tr.S.	780.7	2	390.4	0.70	0.5028
ERROR	19486.5	35	556.8		
Total saturated fat intake					
SEX	528	1	528	1.89	0.1807
TREATMENT	993	2	497	1.75	0.1884
S.Tr.	1025	2	512	1.81	0.1789
ERROR	9923	35	283		
TIME	15.2	1	15.2	0.19	0.6627
T.S.	7.6	1	7.6	0.10	0.7580
T.Tr.	463.8	2	231.9	2.95	0.0652
T.Tr.S.	58.5	2	29.2	0.37	0.6920
ERROR	2748.9	35	78.5		
Total PUFA intake					
SEX	0.572	1	0.572	0.04	0.8380
TREATMENT	94.479	2	47.240	3.50	0.0412
S.Tr.	50.058	2	25.029	1.85	0.1717
ERROR	472.532	35	13.500		
TIME	8.711	1	8.711	1.61	0.2123
T.S.	6.624	1	6.241	1.16	0.2896
T.Tr.	26.548	2	13.274	2.46	0.1001
T.Tr.S.	22.042	2	11.021	2.04	0.1450
ERROR	188.915	35	5.397		

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
Total cholesterol intake					
SEX	32676	1	32676	0.28	0.6006
TREATMENT	334608	2	167304	1.43	0.2532
S.Tr.	325585	2	162792	1.39	0.2624
ERROR	4097581	35	117073		
TIME	1411	1	1411	0.02	0.8859
T.S.	10417	1	10417	0.15	0.6970
T.Tr.	162059	2	81029	1.20	0.3136
T.Tr.S.	85894	2	42947	0.64	0.5357
ERROR	2365267	35	67579		
Blood glucose					
SEX	11.50	1	11.50	0.06	0.8041
TREATMENT	583.81	2	291.91	1.58	0.2194
S.Tr.	119.18	2	59.58	0.32	0.7258
ERROR	6447.06	35	184.20		
TIME	1.354	1	1.354	0.09	0.7616
T.S.	1.062	1	1.062	0.07	0.7881
T.Tr.	59.547	2	29.773	2.06	0.1431
T.Tr.S.	9.685	2	4.842	0.33	0.7180
ERROR	506.775	35	14.479		
Serum uric acid					
SEX	19.9	1	19.9	6.14	0.0181
TREATMENT	4.9	2	2.5	0.77	0.4710
S.Tr.	3.3	2	1.7	0.51	0.6047
ERROR	113.4	35	3.2		
TIME	0.320	1	0.320	1.58	0.2168
T.S.	0.273	1	0.272	1.35	0.2535
T.Tr.	0.359	2	0.180	0.89	0.4203
T.Tr.S.	0.262	2	0.131	0.65	0.5289
ERROR	7.087	35	0.202		
Total plasma cholesterol					
SEX	2696.1	1	2696.1	3.56	0.0676
TREATMENT	448.4	2	224.2	0.30	0.7458
S.Tr.	1136.5	2	568.3	0.75	0.4799
ERROR	26524.9	35	757.9		
TIME	19.40	1	19.40	1.03	0.3177
T.S.	6.30	1	6.30	0.33	0.5672
T.Tr.	83.33	2	41.67	2.21	0.1251
T.Tr.S.	32.57	2	16.28	0.86	0.4309
ERROR	660.88	35	18.88		

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
Total free cholesterol					
SEX	573.1	1	573.1	4.72	0.0367
TREATMENT	562.8	2	281.4	2.32	0.1134
S.Tr.	125.8	2	62.9	0.52	0.6001
ERROR	4249.2	35	121.4		
TIME	26.078	1	26.072	4.85	0.0343
T.S.	0.272	1	0.272	0.05	0.8232
T.Tr.	8.964	2	4.482	0.83	0.4427
T.Tr.S.	0.289	2	0.144	0.03	0.9735
ERROR	188.069	35	5.373		
Total HDL-Chol					
SEX	176.8	1	176.8	3.30	0.0778
TREATMENT	182.5	2	91.3	1.70	0.1967
S.Tr.	76.1	2	38.0	0.71	0.4985
ERROR	1874.5	35	53.5		
TIME	0.527	1	0.527	0.27	0.6089
T.S.	0.005	1	0.005	0.00	0.9593
T.Tr.	1.387	2	0.693	0.35	0.7068
T.Tr.S.	6.081	2	3.040	1.54	0.2294
ERROR	69.283	35	1.979		
Total HDL free cholesterol					
SEX	22.85	1	22.85	0.99	0.3269
TREATMENT	31.91	2	15.96	0.69	0.5082
S.Tr.	75.99	2	38.00	1.64	0.2079
ERROR	809.37	35	23.12		
TIME	1.494	1	1.495	1.41	0.2427
T.S.	0.066	1	0.066	0.06	0.8031
T.Tr.	0.408	2	0.204	0.19	0.8254
T.Tr.S.	3.930	2	1.965	1.86	0.1712
ERROR	37.039	35	1.058		
Total LDL-Chol					
SEX	3627.2	1	3627.2	5.77	0.0217
TREATMENT	1307.4	2	653.7	1.04	0.3640
S.Tr.	2270.8	2	1135.4	1.81	0.1792
ERROR	21993.7	35	628.4		
TIME	6.306	1	6.306	0.58	0.4522
T.S.	0.124	1	0.123	0.01	0.9158
T.Tr.	42.152	2	21.076	1.93	0.1600
T.Tr.S.	7.741	2	3.870	0.35	0.7038
ERROR	381.856	35	10.910		

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
Total LDL free cholesterol					
SEX	353.4	1	353.4	3.65	0.0644
TREATMENT	342.7	2	171.4	1.77	0.1855
S.Tr.	98.6	2	49.3	0.51	0.6056
ERROR	3391.8	35	96.9		
TIME	16.443	1	16.443	6.46	0.0156
T.S.	2.112	1	2.112	0.83	0.3686
T.Tr.	0.399	2	0.199	0.08	0.9249
T.Tr.S.	1.611	2	0.805	0.32	0.7307
ERROR	89.099	35	2.545		
Total VLDL-Chol					
SEX	115.2	1	115.20	1.08	0.3065
TREATMENT	3.4	2	1.70	0.02	0.9842
S.Tr.	25.3	2	12.66	0.12	0.8887
ERROR	3743.7	35	106.96		
TIME	0.802	1	0.802	0.26	0.6155
T.S.	8.249	1	8.249	2.64	0.1131
T.Tr.	2.663	2	1.331	0.43	0.6563
T.Tr.S.	3.074	2	1.537	0.49	0.6165
ERROR	109.296	35	3.122		
VLDL free cholesterol					
SEX	17.35	1	17.35	0.90	0.3499
TREATMENT	1.65	2	0.82	0.04	0.9582
S.Tr.	1.74	2	1.37	0.07	0.9318
ERROR	676.34	35	19.32		
TIME	0.866	1	0.866	1.09	0.3038
T.S.	0.007	1	0.007	0.01	0.9220
T.Tr.	1.686	2	0.843	1.06	0.3573
T.Tr.S.	0.126	2	0.063	0.08	0.9241
ERROR	27.845	35	0.795		
LCAT IRE					
SEX	36.40	1	36.40	4.10	0.0505
TREATMENT	0.91	2	0.45	0.05	0.9501
S.Tr.	14.80	2	7.39	0.83	0.4431
ERROR	310.61	35	8.87		
TIME	2.357	1	2.357	6.54	0.0150
T.S.	0.109	1	0.109	0.30	0.5865
T.Tr.	0.559	2	0.280	0.78	0.4676
T.Tr.S.	1.566	2	0.780	2.17	0.1289
ERROR	12.609	35	0.360		

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
LCAT activity					
SEX	15582	1	15582	8.22	0.0193
TREATMENT	544	2	272	0.14	0.5872
S.Tr.	1106	2	553	0.29	0.7924
ERROR	66361	35	1896		
TIME	268	1	268	3.14	0.3608
T.S.	41	1	41	0.49	0.6542
T.Tr.	107	2	53	0.63	0.5063
T.Tr.S.	364	2	182	2.14	0.9028
ERROR	2991	35	85		

B. Analysis of variance grouping by HDL-Chol

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
Total HDL-Chol					
SEX	191.2	1	191.2	3.57	0.0671
TREATMENT	190.2	2	95.0	1.78	0.1842
S.Tr.	72.8	2	36.4	0.68	0.5132
ERROR	1873.9	35	53.5		
TIME	0.148	1	0.148	0.09	0.7721
T.S.	0.048	1	0.048	0.03	0.8694
T.Tr.	13.122	2	6.561	3.76	0.0331
T.Tr.S.	0.510	2	0.254	0.15	0.8647
ERROR	61.030	35	1.743		
HDL/LDL-Chol ratio					
SEX	0.107	1	0.107	11.96	0.0014
TREATMENT	0.057	2	0.029	3.21	0.0526
S.Tr.	0.056	2	0.029	3.17	0.0543
ERROR	0.312	35	0.009		
TIME	0.000	1	0.000	0.01	0.9405
T.S.	0.000	1	0.000	0.47	0.4965
T.Tr.	0.003	2	0.001	13.52	0.0000
T.Tr.S.	0.000	2	0.000	0.14	0.8734
ERROR	0.004	35	0.000		

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
Total LDL-Chol					
SEX	3529.7	1	3529.7	5.67	0.0228
TREATMENT	1359.5	2	679.7	1.09	0.3465
S.Tr.	2077.8	2	1038.9	1.67	0.2029
ERROR	21771.4	35	622.0		
TIME	1.53	1	1.53	0.15	0.7006
T.S.	1.91	1	1.91	0.19	0.6671
T.Tr.	55.80	2	27.90	12.74	0.0782
T.Tr.S.	29.60	2	14.79	1.46	0.2472
ERROR	355.94	35	10.17		
HDL/Total cholesterol					
SEX	0.018	1	0.018	9.26	0.0044
TREATMENT	0.009	2	0.004	2.15	0.1316
S.Tr.	0.007	2	0.003	1.71	0.1961
ERROR	0.070	35	0.002		
TIME	0.000	1	0.000	0.18	0.6729
T.S.	0.000	1	0.000	0.21	0.6519
T.Tr.	0.001	2	0.000	19.22	0.0005
T.Tr.S.	0.000	2	0.000	0.52	0.5969
ERROR	0.001	35	0.000		
Total serum cholesterol					
SEX	2426.0	1	2426.0	3.20	0.0824
TREATMENT	387.3	2	193.7	0.26	0.7761
S.Tr.	898.0	2	449.0	0.59	0.5587
ERROR	26551.3	35	758.6		
TIME	7.45	1	7.45	0.39	0.5388
T.S.	2.33	1	2.33	0.12	0.7302
T.Tr.	25.22	2	17.61	0.91	0.4117
T.Tr.S.	57.40	2	28.70	1.48	0.2408
ERROR	677.19	35	19.34		
Total activity min/2wk					
SEX	14219	1	14219	0.31	0.5830
TREATMENT	251505	2	125752	2.72	0.0801
S.Tr.	14936	2	7468	0.16	0.8517
ERROR	1620878	35	46310		
TIME	141962	1	141962	10.77	0.0023
T.S.	26271	1	26271	1.99	0.1686
T.Tr.	321787	2	160893	12.21	0.0001
T.Tr.S.	69796	2	34898	2.65	0.0849
ERROR	461229	35	13177		

SOURCE	SUM OF SQUARES	DEGREES OF FREEDOM	MEAN SQUARES	F	TAIL PROB.
Predicted VO2 max					
SEX	861.64	1	861.64	16.39	0.0003
TREATMENT	13.59	2	6.79	0.13	0.8792
S.Tr.	52.92	2	26.46	0.50	0.6090
ERROR	1840.81	35	52.59		
TIME	67.93	1	67.92	60.46	0.0000
T.S.	0.03	1	0.03	0.02	0.8814
T.Tr.	26.06	2	13.03	11.60	0.0001
T.Tr.S.	3.42	2	1.71	1.52	0.2325
ERROR	39.32	35	1.12		
Total calorie intake					
SEX	3299898	1	3299898	8.52	0.0061
TREATMENT	851860	2	425930	1.10	0.3440
S.Tr.	449586	2	224793	0.58	0.5648
ERROR	13549914	35	387140		
TIME	73650	1	73650	0.66	0.4229
T.S.	5622	1	5622	0.05	0.8240
T.Tr.	135446	2	66272	0.59	0.5588
T.Tr.S.	22399	2	11199	0.10	0.9051
ERROR	3919967	35	111999		
Percent body fat					
SEX	546	1	546	6.48	0.0155
TREATMENT	77	2	38	0.46	0.6365
S.Tr.	158	2	79	0.94	0.4000
ERROR	2951	35	84		
TIME	0.20	1	0.19	0.72	0.4021
T.S.	0.03	1	0.03	0.11	0.7472
T.Tr.	0.29	2	0.14	0.52	0.5969
T.Tr.S.	0.12	2	0.06	0.23	0.7955
ERROR	9.54	35	0.27		

XII. APPENDIX F

A. List of abreviations

APO AI: apolipoprotein A-I

APO AII: apolipoprotein A-II

APO B: apolipoprotein B

CVD: cardio-vascular disease

CE: cholesterol ester

HDL-Chol: high density lipoprotein

LCAT: lecithin cholesterol acyl transferase

LCAT IRE: lecithin cholesterol acyl transferase initial rate of esterification

LDL-Chol: low density lipoprotein

LPL: lipoprotein lipase

LPP: lipoprotein

OGTT: oral glucose tolerance test

PUFA: poly-unsaturated fatty acid

STF: standardized test of fitness

SUA: serum uric acid

TG: triglyceride

UC: unesterified cholesterol

VLDL-Chol: very low density lipoprotein

VO2 max: maximum oxygen intake

B. Terminology

Apolipoprotein: a protein with the major function is their ability to stabilize lipids micelles during the transport in blood and chyle; some of the apolipoproteins have been found to have specific physiologic functions as enzyme activators.

Atherosclerosis: a marked deposition of lipids in the inner layer of aterial walls, resulting in formation of elevated fatty fibrous plaques.

Cholesterol: a white, waxy, crystalline organic alcohol; a universal tissue constituent, present in all animal fats and oils, in bile, brain tissue and blood; it is found in deposits in the vessel walls in atherosclerosis.

Foodstyle: refers to quality and quantity of foods intake and also to time repartition of these.

High density lipoprotein: approximately 50% of the HDL-Chol mass is protein (APO AI and APO AII are the major protein), the other major constituents are cholesterol and cholesteryl esters (about 20%) and phospholipids(about 30%).

Lipoprotein: the plasma lipoproteins may be divided into four major classes: chylomicrons, VLDL-Chol, LDL-Chol and HDL-Chol; they have different physical properties in diameter, molecular weight, hydrated density and mobility; they have different chemical properties in protein, triglyceride, cholesterol and phospholipids.

Lipoprotein lipase: is an enzyme that hydrolyses triglyceride contained in the triglyceride rich lipoproteins, chylomicrons and VLDL-Chol. This hydrolysis occurs following the attachement of these particles to the capillary endothelial cells.

Lecithin cholesterol acyl transferase: enzyme which catalyses the conversion of lecithin and cholesterol to lysolecithin and cholesteryl ester. It is assumed that HDL-Chol is the major substrate for LCAT reaction and that virtually all of the plasma cholesteryl esters are formed in the plasma HDL-Chol.

Low density lipoprotein: they transport about two thirds of the serum cholesterol, and in diseases when serum cholesterol is increased, LDL-Chol are usually elevated. Apolipoprotein B comprises approximately 25% by weight of LDL-Chol.

<u>Maximum oxygen intake</u>: the highest oxygen intake that the individual can attain during physical work breathing air at sea level.

<u>Normolipidemic</u>: refers to individuals with normal level of serum lipids including the age and sex factors.

<u>Oral glucose tolerance test</u>: after an overnight fast the subject ingest a pre determined quantity of glucose. Blood glucose and plasma insulin are determined at regular intervals before and for 120 min during the test.

<u>Serum uric acid</u>: in humans, the ultimate catabolite of purines is uric acid. Uric acid is mainly excreted in the urine.

<u>Triglyceride</u>: the basic unit consists of a molecule of glycerol in ester bond with three molecules of fatty acid; it serves as the major storage form of fatty acids and is practically the exclusive constituent of adipose tissue.

<u>Very low density lipoprotein</u>: in the absence of chylomicrons the major portion of the serum triglyceride are transported in this fraction.